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## EDITORIAL

TODAY, even the layman knows that adequate nutrition is fundamental in the treatment of Tuberculosis. There is no need to inform anyone that a properly balanced diet, including vitamins, salts, fats and protein is necessary before healing of tuberculous *foci* can take place. No clinician imagines that an artificial pneumothorax will help the damaged lung unless good nutrition is available to keep up the patient's general resistance. Modern treatment proceeds on the assumption that the patient is receiving this adequate diet. Proper diet is so essential that we take it for granted.

It is unfortunate, therefore, to realise that the tuberculous patient in his own home, as the great majority are, is being left without special dietary care in war-time; his nutritional needs are put no higher than those of any other member of the community. Under the National Scheme of Rationing, he is told to fend for himself.

The Food Rationing (Special Diets) Advisory Committee has advised the Minister of Food that, provided that there is "*an increased consumption of milk and milk dishes, cheese and cheese dishes and eggs*" tuberculous patients need not suffer from lack of first-class protein. Therefore no extra ration of meat was advised. Thus the burden of making up for a tuberculous person's body wastage is thrown upon milk, cheese and eggs. Everyone knows that eggs are not plentiful today, and in some cases unobtainable. They are likely to be even less obtainable as the winter goes on. To many people, cheese, although they are told it is invaluable as a dietary umbrella to protect from omissions, is not specially attractive as a regular article of food. Tuberculous patients often have poor appetites, and the monotonous weeks and months of treatment make them difficult patients to feed. Catering in institutions in the last year was bad enough; the marketing for a tuberculous family is apt to become tragic. It may be doubted whether it is wise and

humane to force tuberculous patients back for their first-class protein solely on cheese, milk and eggs, but since cheese is apt to be monotonous and eggs scarce, the whole emphasis is thrown on milk. If milk is to be his mainstay the patient should at least be able to obtain it in plenty.

Now, if we were desperately short of food and the population were condemned to a subsistence diet, there might be a case for putting the tuberculous person, who is of less productive capacity than a normal individual, on a dietary level corresponding to his value to the nation. But, happily, things are not so bad as that: the Minister of Food tells us there is enough food in the country, that large stocks are in storage, and that further supplies are assured. Those who act on the assumption that the tuberculous person does not require special consideration in these times are illogical, and lacking in knowledge of the tuberculous household.

A new Department of the Government has become interested in Nutrition as a practical issue. The Minister of Food has brought out a new scheme which provides for free milk for mothers and children below a certain income level, and cheap milk for mothers and children of any income level. The response at first was slow, but we understand that now over 80 per cent. of the eligible persons have applied. Though we should have preferred that the Regulations specified "Safe" milk, we wish success to this scheme. It is the fitting outcome of all the Research that has been done in the last fifteen years on the value of milk feeding. There is a very strong case for bringing tuberculous patients under its operation.

It is true that Local Authorities have powers to provide "*extra nourishment*" for tuberculous patients under domiciliary treatment, and this generally includes milk. But the phrase "*extra nourishment*" is interpreted in a vastly different way in various parts of the country, and some Local Authorities make practically no provision of this kind. Others restrict it to cases before and after sanatorium treatment; many allow the issue of milk for a limited period only. One voluntary Care Committee spends £500 a year on milk alone; in certain areas there are no Care Committees whatever. It is no use quoting the example of London or a few places which have well-developed schemes for the prevention of tuberculosis. It is no answer to the demand that tuberculous people should have cheap milk to say they are already catered for by the Local Authorities under "*extra nourishment*" Powers. This is tying red tape round one's eyes. If the theory of "*extra nourishment*" were always carried into practice things would be better; but this is not done, and tuberculous people often fail to obtain the help they need, and to which the law gives them a theoretical right.

Correspondence on this subject has taken place between the Depart-

ments and the National Association for the Prevention of Tuberculosis. Questions have been asked in Parliament, with virtually the same reply. In effect, the official view is: "*All is well. Tuberculous people need not suffer. They have milk, butter, eggs and vitaminised margarine.*" Yet two of these commodities are rationed, and the tuberculous patient receives the same as his healthy brother. Eggs are sometimes un procurable, and the remaining article, milk, is being given free to part of the population but not to the tuberculous adult, though, of course, his wife and children, if he has any, may benefit.

We hope that before long the Authorities will recall the circumstances of our extensive tuberculous population. There are at least 200,000 notified cases in the country, only a fraction of them in institutions. Many have been chronically ill for years and are pitifully poor. Around each tuberculous person is a ring of susceptible contacts. Post-adolescents and young adults are being poured into our industries to help a war effort which protects us all. The argument for fresh measures in the prevention of tuberculosis during the next few years is very strong. There is a cry for more X-ray apparatus and mass screen examinations. We ought not to overlook that the maintenance of proper nutrition is even more fundamental. One of the simplest preventive measures, and an all-round easing of the tuberculous family's plight, would be the extra allowance of milk, free or at a reduced rate, for all adult patients.

We are living at a time when practical nutrition has to be taken seriously by all. Every day we read advertisements from the Ministry of Food broadcasting information on this subject with which doctors and dietitians have long been familiar. The man in the street is learning that the full and balanced diet tuberculosis physicians have always tried to give their patients is now to be his own practical ideal in peace and war. In *Wellsian* anticipations of the post-war world it is taken for granted that everyone will be well fed. It is sad that the tuberculous family should be denied what everyone concedes to the healthy.

In some directions the Minister of Food has shown great vision. Is there any reason why all those interested in tuberculosis should not speak to him on this subject with one voice?

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## GENERAL ARTICLES

### ROUNDING OUT THE TUBERCULOSIS CAMPAIGN

By LOUIS I. DUBLIN,

PH.D.,

Third Vice-President and Statistician, Metropolitan Life Insurance Company, New York.

IN general, the results of the campaign against tuberculosis in the United States have been most gratifying. The death rate from the disease has dropped steadily for three decades and even depression conditions have not slowed up or even halted the downward trend in most areas of the country. The current death rate in the general population, of about 45 per 100,000, represents a decline of over 70 per cent. in the last thirty years. Among the millions of Industrial policy holders of the Metropolitan Life Insurance Company, who reflect the trend in the wage-earning population, the decline in the tuberculosis death rate in the past thirty years has been about 80 per cent. These are achievements which not even the most optimistic observers a generation ago would have thought possible to attain in so short a time, and give great encouragement to the idea that tuberculosis can be practically eradicated in this country in the next two decades if we correct the chief weaknesses in our campaign against the disease.

Many forces have been at work to bring about the great improvement in the mortality from tuberculosis. In great part, the improvement reflects the gradual rise in the general standard of living which has taken place in this country; but the efforts of private anti-tuberculosis agencies and of public health workers have been powerful influences also. The tuberculosis clinic and its auxiliaries have been developed to a higher degree in America than anywhere else in the world. Through their operation, an undoubted check was given to the spread of tuberculosis in the families of patients. Continued and intensive public health education among both children and adults has taught to increasing numbers of people the symptoms of tuberculosis and the way in which the disease spreads. Much work has been done in the development of sound case-finding methods, which are being given increasingly wide application. Finally, the extensive develop-

ment of hospitals and sanatoria for the domiciliary care of the tuberculous has likewise checked the spread of the disease to others and at the same time has sent back truly huge numbers of persons to their communities with their disease arrested.

Those of us who have laboured in the field of tuberculosis have every reason, therefore, to be gratified with the results achieved. There is no room, however, for smugness or self-satisfaction. Our programmes for the control of the disease have, for one thing, not been as well balanced as they might, and in certain respects have been woefully inadequate. This applies particularly to our case-finding methods, and even more to our provisions for the rehabilitation of the tuberculous. If these two phases of the programme had been developed adequately, our national figures would have been much better than they are.

The relatively poor progress in our case-finding methods is attested by data on condition of patients at admission to sanatoria in 1938, according to a recent report of the Council on Medical Education and Hospitals of the American Medical Association. This study showed that for sanatoria in the United States as a whole, only 12·9 per cent. of the patients with pulmonary tuberculosis of the re-infection type were admitted in the minimal stage, whereas 32·1 per cent. were moderately advanced at admission and not less than 55 per cent. far advanced. These figures are almost identical with those in a similar report of the Council made on the basis of 1934 data. It is not surprising, therefore, that, according to the earlier of these reports of the Council, at discharge only 17·2 per cent. were arrested or apparently arrested, and 11·6 per cent. quiescent, while 30·1 per cent. were "improved", 17·2 per cent. unimproved and 23·9 per cent. died at the sanatorium. Such results are hardly commensurate with the excellent facilities of these institutions or with the huge capital invested in them. It should interest British readers that approximately 100,000 beds are now available for the care of the tuberculous in the United States, and that these represent a capital outlay of over 300 millions of dollars and annual maintenance costs of approximately 80 millions of dollars.

Even more reprehensible is the failure to see the problem of the tuberculous patient as a whole or to co-ordinate efforts in his behalf from beginning to end. Too often the results of good case-finding are made futile by inadequate treatment, because facilities for treatment are over-taxed, and patients not kept at sanatoria long enough, or because the patient, worried about the distress of his family arising from the loss of his wages, leaves the sanatorium too early. But even when this obstacle has been overcome, the patient, fresh from the sanatorium, attempts to resume full-time work although he is usually incapable as yet of standing up to the same level

and intensity of physical activity as his fellow-workmen. How often, also, is the useless advice given him to seek new outdoor employment which often requires a degree of physical exertion to which, even before the onset of his tuberculosis, he was unaccustomed. But perhaps worse than such meaningless advice is the lack of interest in and lack of effective guidance for the sanatorium graduate when he most needs it—when he is faced with the problem of adjusting himself to the real world on his return home. The rehabilitation of the tuberculous is the weakest link in the American anti-tuberculosis campaign.

This unfortunate situation is readily amenable to marked improvement, as is indicated by the experience of certain sanatoria, serving certain special groups who are under continuous medical observation. Perhaps the best example is offered by the experience of the Metropolitan Life Insurance Company with respect to tuberculosis among its employees. The programme of this Company is conceived on a very broad basis covering not only the detection of the disease in applicants for employment and among active employees, but sanatorium treatment until the employee is able to resume his former occupation, and careful medical supervision after his return to work. Moreover, through his disability insurance, the patient usually receives a good part of his income during his sanatorium stay. Since his family is thus provided for, he is the more willing to remain until his disease is arrested. The detection of the disease among employees is facilitated by medical examination, annually or oftener when necessary, of all persons in the service of the Company. At the Home and Head Offices, furthermore, fluoroscopic examination of the chest is routinely made as part of the annual examination and after illnesses, particularly respiratory disease and repeated colds. This technique has proved highly effective in discovering cases with abnormal chest findings, for whom roentgenograms are advisable.

As a result of this case-finding programme, a high proportion of the persons, connected with the Company, who have developed tuberculosis have been detected while their disease was still in the minimal stage. This is particularly true of the Home Office personnel since 1928, when routine fluoroscopy was instituted. In that group, in fact, a high percentage of those developing tuberculosis have been detected before there were any clinical signs or symptoms of the disease or any appreciable loss of weight. Moreover, the incidence of new cases of the disease has definitely declined, both among Home Office employees and Field representatives, although the incidence of the disease among applicants for employment at the Home Office, who are drawn pretty much from the same population as those actually employed, has shown no significant decline. In the last ten years

alone, the reduction in the number of new cases of the disease at the Home Office is nearly 80 per cent. Even with this large decrease in the total of new cases from the Home Office, those with minimal tuberculosis on admission to the sanatorium comprised 59 per cent. of the total during 1930-38; 36 per cent. moderately advanced, and only 5 per cent. far advanced.

Adequate sanatorium care is the second essential in the Company's programme of rehabilitation of the tuberculous. To meet that requirement, the Metropolitan's sanatorium aims to keep its patients until they are ready to resume work. This sanatorium is an up-to-date and well-maintained group of hospital buildings located at Mount McGregor, New York, nine miles north of Saratoga Springs. The sanatorium is situated about 1,200 feet above sea-level. Established late in 1913, it has been expanded by stages to a bed capacity of 350 beds, although in recent years, with the decline in the number of tuberculous employees, a large part of its bed capacity is devoted to the treatment of non-tuberculous conditions.

The objective of keeping tuberculous patients until ready to return to work cannot always be achieved, of course, because a fairly large number of patients, for various reasons, leave the sanatorium before they complete their cure. Most of these patients feel well, but cannot by accepted clinical standards be called arrested. A few also who are not doing well want to spend their last days with their families. Yet, of the 1,069 patients first admitted for treatment of tuberculosis between 1919 and 1936, and discharged by the end of 1936, 55.2 per cent. were in a satisfactory\* condition at discharge. Those discharged as "improved" accounted for 23.2 per cent. of the total; 13.9 per cent. were discharged unimproved; and 7.7 per cent. died at the sanatorium.

How important the efforts at early detection of tuberculosis are can be seen in a comparison of the results at the Company's sanatorium among minimal cases, as compared with advanced disease. Of those admitted with minimal tuberculosis, 71.5 per cent. were discharged in a satisfactory condition, as compared with 51.5 per cent. of the moderately advanced and 32.9 per cent. of the far advanced. Furthermore, only 1.1 per cent. in the minimal group died at the sanatorium, as compared with 6.7 per cent. in the moderately advanced group and 22.8 per cent. in the far advanced group.

This policy of keeping the patient at the sanatorium until he is fully

\* Arrested, apparently arrested, or quiescent. Those quiescent at discharge from the Company's Sanatorium are included because only nine of the 250 patients in this group had a positive sputum at discharge. This is a much lower percentage than at most other sanatoria.

ready to take up his former job is justified by the excellent long-term results obtained. If we consider only those patients discharged alive who, in 1937, had been out of the sanatorium at least five years, 61·7 per cent. were at work or able to work on the fifth anniversary of discharge. Of the remainder, 18·3 per cent. were unable to work, usually because of relapse, a few had retired because of age, 3·0 per cent. were known to be living but their ability to work was unknown, and 15·7 per cent. had died. Only a few patients were untraced.

The effect of early detection of the disease is seen also in these long-term results. Of those with minimal tuberculosis on admission who were discharged in satisfactory condition, over 80 per cent. were at work or able to work five years after discharge, only 10 per cent. were unable to work, and 3·5 per cent. had died. The mortality of this large group of people was only slightly higher than that among their non-tuberculous fellow-workers. These figures are in marked contrast to those with far-advanced tuberculosis who were in unsatisfactory condition at discharge. Of this group, less than one-fourth were able to work on the fifth anniversary of their discharge, about one-half had died, and most of the others were ill or unable to work.

In the experience of this life insurance company, then, we have a striking example of the effectiveness of a programme of early detection of tuberculosis, combined with adequate sanatorium care and, equally important, subsequent provision of work suited to the individual. Moreover, medical supervision of the ex-patients after discharge from the sanatorium brings about early discovery of a threatened relapse. How different is all this from the experience of most sanatorium patients who are concerned over their families and who, on discharge, suffer the mental distress involved in finding new and profitable employment.

Quite different in method, if not in aim, is the rehabilitation work carried out under the auspices of Altro Work Shops, Inc., of New York, which is conducted by the Committee for the Care of the Jewish Tuberculous. Naturally a private agency of this type cannot do much in the way of case-finding beyond the contacts of the persons it serves—namely, those already diagnosed as tuberculous. But within its field, the breadth and excellence of the Committee's programme are seen in this statement of its purpose: "The man or woman with tuberculosis must be considered (1) as a sick person to be restored to health, (2) as a member of a family, with that family's particular problems and limitations, (3) as a potential worker, to be restored, in so far as is possible, to his place in the community." The Committee recognised also the interdependence of these various aspects of rehabilitation.

The Committee developed workshops in which former sanatorium patients work according to their capacity, in good surroundings and with proper provision for rest as necessary. Thorough medical supervision is an important part of the set-up. The Altro Work Shops are located in New York City, although outside the congested factory area. It manufactures garments of various types, particularly those needed by the personnel of hospitals and hotels. The Committee chose the garment industry for its field because so many of the patient group with which it dealt had formerly worked in this industry. The shops are conducted entirely on a business basis. The merchandise is sold both through mail and direct sales. The property of the shop represents an investment of approximately \$190,000. The equipment and inventory represents a further total of over \$80,000.

Each worker at the Altro Work Shops is paid at piece rates. The Committee, however, supplements his actual earnings by a subsidy which varies according to the needs of the patient's family.

The success of Altro is shown by both its medical and financial results. Of the 304 patients first admitted to Altro in the ten-year period ending in 1924, 66 per cent. were well and able to work six months to ten years (average five years) later. Of the same group, after an additional ten years, just about half were still well and working. Five per cent. were living but unable to work, 28 per cent. had died, and 18 per cent. were untraced. Of a more recent group of 502 patients admitted to Altro between 1925 and 1934, 80 per cent. were well and working six months to ten years later, 9 per cent. were living but unable to work, and 10 per cent. had died. It is notable that an increasing proportion of patients with advanced tuberculosis has been taken on in recent years.

Of distinct interest are the results on 66 patients with positive sputum, admitted to Altro during 1927 to 1936. Of this group, 61 per cent. were well and working six months to ten years (average six years) later.

The financial side of this venture is worthy of brief mention. During a little over twenty years, Altro workers have received nearly one and one-half million dollars in actual wages, exclusive of subsidies described above. The total operating loss sustained by the organisation in this period was \$180,000.

In short, the Altro patients have done well not only medically, but they have given a good account of themselves economically. It is not long before they become, to a large degree, self-supporting. It cannot be too much stressed that the amount of the subsidy, used in the rehabilitation of these patients and their families, is only a small part of what is usually required from charitable or municipal sources to maintain patients in

sanatoria and their families at home. The living standards of Altro and similar families are, moreover, incomparably better than are the beneficiaries of the usual type of charity.

I can reaffirm here what I said on the occasion of the twenty-first anniversary of the founding of the Altro Work Shops:

"The real point I wish to make to-day is that the experience of the Altro Shops, of the Metropolitan employees and of the workers in the industrial villages of England, must be generalised. The facilities for social rehabilitation of the tuberculous in the United States are pathetically inadequate. Only about 1 per cent. of those who are discharged from sanatoria receive the benefits of the kind of after-care that we have been describing. It is necessary to make available to sanatorium graduates uniformly this type of rehabilitation. It is obviously folly to continue to spend a hundred million dollars for the care of the tuberculous as we do each year, only to cast these patients adrift at a time when they most need supervision and guidance. The experience of your organisation must be made the guiding star for every worthwhile tuberculosis institution. A sanatorium which has no connection with an industrial agency is to that extent a limited facility and deficient to that degree. The time has come when industrial rehabilitation must be an inseparable and integral part of the restoring process and of the re-establishment of the tuberculous patient as a member of the community. We must now tell the whole world how vital and significant has been the experience of this institution, whose coming of age we celebrate to-day."

It is my definite belief that if we round out our campaign against tuberculosis by placing increased emphasis on case-finding and rehabilitation of the sanatorium graduate, we can, within the next twenty years, bring the incidence and mortality from tuberculosis to a point where it is a comparatively minor factor in the disease picture. For this purpose we shall, of course, have to increase measurably our personnel and our medical facilities. We shall be wise, furthermore, to direct our new efforts in more carefully selected directions than in the past. We need to concentrate on such groups as manual labourers and their families, especially negroes, on workers in mines and factories where there is a specific hazard from silica dust, on young women of childbearing ages, and, with respect to case-finding, on the older persons with a chronic cough. These are the principal reservoirs of new cases requiring attention. Fortunately there is a clearer understanding to-day than there was hitherto of the shortcomings of our tuberculosis campaign, and there is greater readiness to remedy these defects. The cry to improve case-finding has not gone unheeded. Efficient and economic methods are now available to make easier the administrator's

job. Again, thanks particularly to British workers, as well as a few pioneers in this country, there is already widespread appreciation of what constitutes a sound programme of rehabilitation. It is, therefore, no longer fantastic for some of us who were active in the initial stages of the anti-tuberculosis campaign in America to expect that we shall witness the final stages as well. That will prove to be an event well worth the effort of a lifetime.

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## REHABILITATION

By SIR PENDRILL VARRIER-JONES,

F.R.C.P.,

Director, Papworth Village Settlement.

IT is axiomatic that a machine cannot function with efficiency if it lacks essential parts. An aeroplane will not fly, for example, if it lacks a rudder and has no wings. For a limited purpose—to travel noisily but aimlessly along the ground—it may be a great success. But as an aeroplane it is a failure.

In exactly the same way the anti-tuberculosis scheme fails because it lacks essentials. It is impressive so far as it goes; but it does not go far enough in either direction. It is all middle, and no ends.

The two missing essentials are, first, early cases; and next, rehabilitation.

It is not too much to say that if the first essential were present, the last would be unnecessary. But as the first is missing, and the second undeveloped, the whole scheme is rendered inadequate. I hope in the ensuing pages to show that I do not exaggerate and that there is no reason whatever why this inadequacy should persist.

THE BRITISH JOURNAL OF TUBERCULOSIS of October, 1932, contained the following significant statement (p. 163): "In the first place, the vast majority of students qualify and enter practice without seeing possibly a single case of early infiltration. They are thus unable to detect pulmonary tuberculosis at its onset."

This is very true. How many of us who have long emerged from the student stage can really "detect pulmonary tuberculosis at its onset"? This is the probable explanation of cases of tuberculosis being accepted for the fighting Services and later discharged with marked lesions.

In my address to the Congress of the Royal Institute of Public Health

held at Eastbourne in 1933, I ventured to develop this point. I claimed that in point of fact we do not know, and cannot as yet determine, the moment of onset (by which I do not mean the moment of infection); and I suggested that we ought to extricate ourselves from the "morbid anatomy groove" and direct our attention towards the vitally important question—the moment of true onset. "A new definition is needed—not the old idea that onset was contemporaneous with the discovery of symptoms, but the newer conception, that onset is the moment at which the equilibrium of the defensive mechanism is first threatened."

So far as I can ascertain, almost no notice has been taken of this suggestion. We are still willing to refrain from diagnosing tuberculosis until the clinical signs are so clear that the case has ceased to be early. And indeed we can hardly be blamed for that. To make a false diagnosis, to scare the wits out of a patient by suggesting tuberculosis when in fact none is present, is not the way to make progress in medicine. In consequence we wait and go on waiting, in most cases until definite sputum or X-ray evidence is available to confirm our suspicions.

Now is it not certain that something must happen inside the patient before the tissue changes are sufficiently considerable to cast a shadow in the X-ray? Surely, therefore, it is not tissue destruction or fibrosis which we should study, but the causes thereof. It is useless to study effects while ignoring causes; and the X-ray shadow is an effect, not a cause. Something has caused that shadow. Very well. What? The entrance of tubercle bacilli into the lung? Perhaps; but those bacilli enter the lungs of 85 per cent. of the entire population, in *most* cases producing no clinically observable effect whatever. That, obviously, is an insufficient explanation of the fact that in *some* cases those bacilli cause a tremendous stir. Mere infection alone cannot and does not explain the matter. What needs explanation is this very fact, that in *most* cases an invasion of tubercle bacilli leaves the host unmoved, whereas in *some* cases it starts a disease process which has a fatal issue.

Surely the problem really revolves around the degree of tuberculous toxæmia which is set up by the invasion. There is fairly general agreement on this point; but there is still no satisfactory explanation as to why an invasion sometimes creates a toxic condition so acute that disease results, whilst in the majority of people it does nothing of the kind.

I am aware of the term "diathesis". I know that there is such a thing as "natural resistance". What I do not know is how to translate these admirable words into practical biological or biochemical terms. There is tremendous variation in the "natural resistance" of individuals. *Why?* That is the point. Are we not driven to the conclusion that we are as yet

almost entirely ignorant of the action and interaction of the tissue fluids and internal secretions which determine the degree of resistance to the tuberculous toxæmia ?

When we have devised a yardstick by which we can measure or define these exchanges, we may be within sight of the first recorded early case. Hitherto few, if any, so-called "early" cases have been truly early; and the sooner this depressing fact obtains general recognition the sooner we shall begin to make some progress in the sphere of prophylaxis.

Not content with the unfortunate error of describing, as "early", cases which—as we can now see—are not early at all, we have constructed a vast system of institutional and dispensary treatment upon this altogether false and misleading foundation. Thousands of beds in our sanatoria are filled with cases which are anything but early. Many are admittedly "advanced"; more still are "moderately advanced". Sanatoria were never intended for such. They were intended, quite rightly, for "early" cases; and we have quickly placed the cart before the horse. Instead of concentrating upon defining and then discovering truly early cases (a matter requiring a great deal of observation and study), we have thrown ourselves eagerly into the far easier but infinitely less valuable task of building bigger and better sanatoria, as though architecture could solve our problems for us.

What is the result? The sanatoria are crammed with cases which they cannot hope to cure. In they go; then out; in again; out again; over and over again. It is a game of cat and mouse, a cruel game and a wasteful one too. Some of these chronic cases cost the community as much as £1,000 each in treatment fees, and after all the expense the patient is alive but demoralised. For years he has been, not a person, but a Case; and it is a dreadful thing to live for years as a Case.

It is not natural to anyone to be denied a means of self-expression, to live in a condition of chronic frustration. Such a life is a living death. It saps the moral fibre and ruins the personality. Some years ago *The Lancet* emphasised this in a valuable article insisting that a constructive outlet must be provided for the "aggressive impulse". Aggression is unpopular just now; but the impulse is present in greater or less degree in everybody, and its prolonged suppression undermines the personality and converts even the most human of beings into nothing more than a Case.

We have admitted many such Cases to Papworth. They have been in and out of sanatoria for years. When they come to us they find in our Industries "a constructive outlet for the aggressive impulse". Some of them are too demoralised, too lacking in moral fibre, to take advantage of this. They, unfortunately, have become Cases beyond hope of redemp-

tion. Others slowly recover their personality and in time take their places as useful members of our community. This destruction of moral fibre is undoubtedly a fact which should engage more attention than it does at present. It is one of the most distressing features of the sanatorium system and it is bound to be imperfectly realised so long as there are physicians who make the mistake of concentrating upon the damaged organ rather than upon its host. Treat the patient, the whole patient, and his environment as well, and *he* and his body fluids will conquer his distemper far more surely than any quantity of medical, surgical and X-ray apparatus.

If, in spite of the disadvantages indicated, the anti-tuberculosis scheme succeeded in protecting the general community from tuberculosis, one might tolerate the sacrifice of the few for the benefit of the many. But are we even giving this protection?

The answer is in the negative. To begin with, there are in this country fewer than 30,000 institution beds for consumptives; but there are 70,000 sputum-positive cases, to say nothing of more than 200,000 other consumptives on the dispensary registers. Thus there must always be 40,000 positive cases "at large". The corresponding figure for the United States in 1937 was 57,000. What sort of protection is this? And what would the public say if they realised the position? Imagine the outcry if there were 40,000 lepers "at large". *Truth* and the *Sunday Express* have recently been engaged in a battle of words on the subject of a single alleged leper amongst the prisoners rescued from the *Altmark*; but neither journal, so far as I know, has discovered whether there were any consumptives on board, although tuberculosis is quite as fatal as, and—according to *The Lancet*—"perhaps more infectious than leprosy"!

Where are these 40,000 sputum-positive patients? In their homes, in the streets, in the buses, tubes and trams, in railway carriages, in schools, everywhere. Most of them are looking for work. Some lucky ones find it.

Two of these lucky ones wrote to Papworth the other day. Both had jobs; both feared a breakdown. One was employed in a preparatory school. The other was in constant contact with persons in impaired health. Both would be sacked instantly if their condition were known; *but it isn't*.

With such instances coming to notice day after day, can we even pretend that we are trying to eradicate tuberculosis?

It is wrong for us to make any such pretence. No one conversant with the facts would believe us if we did. And it is useless for the medical profession to deny responsibility. We are responsible. The public rely upon the tuberculosis service, for which they pay, to protect them against the disease, just as they pay for the armed Forces to protect them against the nation's enemies. What would the public say to the Imperial General

Staff if it tolerated 40,000 spies in our midst? A great deal; so much indeed that there would soon be a new General Staff and probably a new Government as well. Yet the anti-tuberculosis service tolerates 40,000 sources of infection, each—as was well said in *The Times* by the National Association for the Prevention of Tuberculosis—more potentially dangerous to life than the average bomb. Can there be any doubt but that we are failing most grievously in our public duty?

The truth is that we owe an apology to the public. We have set up, at their expense, a widespread organisation perfectly adapted to the treatment and “arrest” of early cases, and we have completely failed to discover the early cases. The scheme looks so good, however, and the sanatoria look so fine, that we are reluctant to admit that the results are disappointing. Indeed, we often protest that they are not disappointing at all, and claim that as the death-rate is falling this phenomenon must be due to our scheme.

Again and again one sees this argument put forward, and it is high time it was challenged. If the argument is fallacious it cannot any longer be used to justify the expense of our semi-complete scheme; and fallacious it most certainly is.

I quote from *Tuberculosis, Cancer and Zinc*, by Dr. D. B. Cruickshank (Medical Publications Ltd., p. 20): “A study of the death-rate statistics reveals that many years before the introduction of anti-tuberculosis campaigns the death-rate had commenced to fall. Moreover, the later introduction of available scientific treatment and control of the disease has had not the slightest effect on the death-rates. The death-rate has continued to fall at a practically constant rate, apparently quite independently of any interference from without. For example, the discovery of the tubercle bacillus, the causative agent, and with it the much more enlightened and rational therapy and preventive measures which this knowledge made possible, has equally failed to influence or in any way accelerate the steady decline in death-rates.”

From this it follows that our scheme in its present form, unsupplied with early cases and unsupported by comprehensive rehabilitation, is little better than a placebo. It has deceived all of us, and hypnotised us by its size and cost into imagining that it is a great success, whereas in fact it is nothing of the sort. Something else—something quite other than our scheme—has sent the death-rate down; and we ought never again to give credit where it is not due.

Now the last thing I wish to suggest is that our sanatoria themselves are wrong, or badly run, or unnecessary. Nothing is further from my mind. My point is that we have set our sanatoria an impossible task. It would be ridiculous to blame them, or those in charge of them, for failing to achieve

the impossible. The responsibility for the existing failure lies, not upon the sanatoria, but upon ourselves who pretend that the impossible is in fact being achieved, and who by means of this pretence prevent any remedy from being applied upon the comprehensive scale which the size of the problem demands.

Surely the facts are that the dispensary and the sanatorium are essential but *central* items in any anti-tuberculosis scheme; that if they were used for early cases only they could reduce the tuberculosis mortality to negligible proportions; that without early cases they can achieve little; and that as they do not get the early cases they must be supported by an after-care scheme which will meet and defeat by means of rehabilitation the complex of psychological, social and economic problems which beset the average patient after active and prolonged sanatorium treatment has done its part.

What is rehabilitation? The Oxford Dictionary defines it as "the action of re-establishing in a former status or standing with respect to rank or legal action". It would be interesting to know just how many tuberculous persons are ever rehabilitated in accordance with this definition. A number of rich ones may be, if they are not obliged to work in order to live. But the tuberculous worker is rarely rehabilitated. He is far more often ruined. Even if he does not become a Case it is unusual for him to be re-established in his former status; and it is just because this is generally known and recognised by the public at large that laymen will go to any lengths, not to obtain treatment, but to avoid it. *They do not want to be ruined.* They do not understand anything about infiltrations, diatheses, lesions, and so on. They are shockingly ignorant of the rôle of the bacillus and of heteroallergy in tuberculous liquefaction. Indeed, they are not interested. What they do understand quite clearly is that if they are labelled T.B. they will ten to one lose their jobs and stand little chance of getting others. That is why so many new cases are already moderately advanced before discovery, while in some 4,000 cases a year death even precedes notification.

It is no comfort to these people to tell them that after treatment they will be fit for work, that most of their working capacity will be restored. Working capacity is useful *only* to the extent to which it can be converted into wages, and, as every layman and ex-sanatorium patient knows, it is one thing to possess working capacity, but quite another to sell it—*i.e.*, to exchange it for a salary or wages. Medical men may not perhaps realise this. They are not trained in such matters. Their training is on a pathological rather than a practical basis; and I often think that it would be an excellent thing if no student were accepted at any hospital until he

could prove that he had earned his living for at least one year in some lay occupation. In that year he would learn nothing about biology, but he would learn a great deal about life. This may not seem important or desirable to some; but in my view it is both, and especially so in relation to tuberculosis.

Be that as it may, we are confronted by the urgent necessity of either discovering truly early cases and thus enabling the sanatorium-dispensary system to function with success; or of establishing rehabilitation upon a scale which will command public confidence. It is not enough to establish little schemes here and there. It is not the medical profession which must be impressed, it is the general public; and not until the general public have confidence in the size and scope of measures for the rehabilitation of the tuberculous will they cease to regard tuberculosis treatment as though it were more terrifying than the disease.

*The Lancet* (January 7, 1939), in a leading article stated that "the average consumptive leaves the sanatorium in a state which is little better than an equilibrium between attack and defence. The patient who just holds his own while putting no strain on his resources relapses directly he is compelled to draw upon them. The credit in the account of the consumptive is always a very small sum."

These words should be illuminated and framed and placed on the desk of everyone who is a consenting party to the cat-and-mouse game previously described. They are as compelling an argument for large-scale rehabilitation as can be found anywhere. The problem is a large one, and it involves many non-medical factors. It is primarily medical; but it is compounded of almost equal parts of sociology, industrial management, economics and general administration.

Thus it is entirely right and proper that Lord Willingdon, whose experience of men and affairs is unquestionable and unquestioned, should have an opinion on the subject; and in his article in *The Times* of January 13 last he expressed himself forcibly on the subject.

Denouncing the "waste arising from accidents and disease, and in particular from tuberculous disease", Lord Willingdon remarked that "large sums are being spent upon the treatment, and even upon the subsequent training, of sufferers; but although medical science has achieved wonders in restoring working capacity impaired by sickness or accidents, there is no national scheme for converting that restored capacity into wages. Most of the value of the restorative work is thereby wasted. No one really knows the extent of this waste, or the annual cost in cash and suffering. But there are 300,000 persons on the tuberculosis registers and 380,000 workpeople are said to be injured each year in industry. The

annual cost to industry of accident compensation has been put as high as £6,000,000 sterling, and even these large figures take no account of the disablement arising from road accidents or from war."

The figures are impressive; and after discussing the respective merits of State and voluntary bodies as the media through which rehabilitation on a national scale might be achieved, Lord Willingdon rejects both.

"If neither State control nor the voluntary system will meet the case, therefore, what alternative is there? Surely it is to be found in a memorandum submitted to a Government Committee in 1936 by Sir Pendrill Varrier-Jones and Mr. Reynell Wreford. It was there suggested that a National Rehabilitation Board should be constituted 'to operate as a public utility on the lines of the Electricity Board'. The income of this board, according to this memorandum, would be derived from the sources which are at present dispensing unemployment and health benefits. This proposal is not new in principle. It was first put forward by the same authors in 1929, and again, more formally, in 1930, when it was submitted to the Ministry of Health. It suggested that if the public and private bodies concerned were given the statutory power to divert their relief expenditure in the case of disabled persons to the service of rehabilitation loans, the whole scheme would be placed immediately upon a sound foundation. Commenting upon these proposals in August, 1930, the Official Gazette of the County Councils Association declared that 'the scheme has the advantage of not adding one penny to present expenditure upon health and social services and is worthy of the closest consideration'. It would appear, therefore, that the idea is a practical one. The functions of the board would be to provide, upon a national scale, the link between working capacity and the opportunity to earn. With this in view, it is suggested that the board should gradually become the Contractor-General to the public authorities. By this means the disabled would be converted from a liability into an asset, and the ultimate saving would be enormous. The benefit of this saving would be felt by every ratepayer and taxpayer, by every industrial insurance company, by the trade unions, and by the friendly societies. And the benefit of the scheme to the disabled themselves would be incalculable. Instead of being unemployed, they would be employed, at union rates, under conditions suited to their disabilities."

This view found favour with the leader-writer of *The Times*. "The time has come," he wrote in the same issue, "to profit by experience in respect not only of tuberculosis but also of other crippling diseases. A great burden of anxiety and distress must be borne by those who have been deprived of the means of earning a living; and that burden is necessarily shared by the community in which they live. Very large sums of money

are being spent upon institutional treatment and upon treatment at home, but very small results have so far been achieved; nor does it appear that substantial improvement can be expected from these methods. What is necessary is rehabilitation—the power to earn exercised in circumstances calculated to maintain and to secure that power."

Almost immediately after the appearance of these articles *The Lancet* returned to the charge on January 20, 1940, with another leading article. After generously describing the Papworth scheme as "the most comprehensive single effort to deal simultaneously with every aspect of the tuberculosis problem" which "saves more than it costs", it cordially supports Lord Willingdon, and concludes: "The beneficent possibilities of the scheme are indeed endless, and bearing in mind that it would not only eliminate much waste and misery, but also mobilise the unfit in the interests of the nation, we hope that the Minister of Health will lose no time in conferring with the Papworth authorities and with the various other interests concerned."

Many details require to be resolved: the personnel of the proposed Board; the mechanism whereby the unproductive expenditure shall be diverted; the rate of interest to be paid; and so on. It would ill become me to anticipate any decisions which may in due course be taken, and I have no intention of doing so; but in view of the fact that both *The Times* and *The Lancet* have lost no time in suggesting that the scheme would be beneficial to persons disabled otherwise than by tuberculosis it would evidently be unwise to assume that the matter will lie wholly within the purview of the tuberculosis service. We shall be consulted, no doubt. Rehabilitation is at present more essential in connection with tuberculosis than in connection with probably any other form of disability. But this may not always be the case, and if proper use be made of a big rehabilitation scheme it may not be the case for more than a very few years.

For is there any field which provides the same unique opportunity for studying the mechanism of resistance as a village settlement? Where else can be found hundreds of tuberculous persons, *and their contacts*, living under observed conditions and in a known environment, not for months but for years at a time? Give us rehabilitation; give us this chance of studying the mechanism of resistance at close quarters; and if we cannot before very long define and discover a really early case it will be very remarkable indeed. So far as resources permit we are engaging in this study at Papworth. But Papworth is neither rich enough nor large enough to produce incontrovertible evidence on the vital subject of true onset. We need a wider but similar field; and if then we fail to find what we seek it will be both surprising and disappointing.

Thus I submit that if we are honest, if we are sincere, in our efforts to eradicate tuberculosis we must face the facts and discard our pretences. We must provide the second essential—rehabilitation—for our cases, both for the sake of those cases and for the sake of the public whom we are failing to protect. And having secured rehabilitation, we must use to the utmost the wonderful opportunities which it will provide in order to discover the first essential—the truly early case, which will be curable instead of chronic, and which will give our sanatoria their first real chance of proving their capacity.

If we will only do this, if we will only unite to press this view upon High Authority, the thing will be done. We must press for a rehabilitation board. On the basis suggested it will add not a penny to the public charge. Having got it, we must use it for all we are worth; and then, our own problem solved, we may be able to turn it over to the rheumatics and the heart cases, to the fractures and the war disabled, to whom it will ever prove a necessary and invaluable friend.

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## MENTAL DISTURBANCE IN PULMONARY TUBERCULOSIS

By R. Y. KEERS,

M.D.,

Medical Superintendent, Tor-na-Dee Sanatorium.

EVERY sanatorium physician is fully aware of the immense importance of the mental attitude adopted by the patient towards his disease and the bearing which this has on both the immediate and ultimate prognosis. In fact, its importance cannot be over-emphasised; and on the patient's ability to adjust himself to the changes in his life engendered by a chronic and disabling disease in which the economic factor plays a major rôle, his future well-being depends. In the majority of cases these adjustments are satisfactorily negotiated, in others the result is achieved only after much toil and travail on the part of both patient and doctor, while in a small group we are confronted with a definite failure. The lot of this last section is a hard one both mentally and physically.

The question of the interrelationship of definite psycho-pathological manifestations occurring in the course of tuberculosis has given rise to discussions at various times in the literature, and the precise connection between the two processes has been debated at some length.

T. S. Clouston noted that the death rate from pulmonary tuberculosis in the Royal Edinburgh Asylum during the decade 1879-88 was higher than that of the general population in the ratio of 3 to 1; and Silk (1917) reported that 71 per cent. of cases of dementia praecox which had come under his observation developed pulmonary tuberculosis. This latter observer, however, was of the opinion that the tuberculous process followed on the mental disease as a result of the lowering of the patient's vitality, and he expressed the view that tuberculosis was never a direct cause of insanity, although in certain cases it might act as a proverbial last straw, where an individual's adjustment to his environment depended on a fine balance. This opinion is supported by the conclusions of Forster and Shephard (1932), who reported the examination from the psychiatric standpoint of 100 cases at Cragmoor Sanatorium. They found four cases of psychosis, but could establish no essential difference between these and cases of psychosis occurring in the general population. Bannister (1930) also expresses the view that there are no specific psychoses or psychoneuroses connected with tuberculosis as a direct consequence of the disease.

The following case histories are presented, showing varying types of mental disorder which were encountered in a private sanatorium during a period of seven years and the part played by the tuberculosis in their aetiology.

CASE 1.—A married woman aged forty was admitted to the sanatorium on June 22, 1932. Her previous history was uneventful, apart from an attack of pleurisy with effusion on the left side nine years earlier. On admission X-ray examination showed an area of infiltration in the left upper zone, without any evidence of cavitation. She was toxic, the blood sedimentation rate was much accelerated, and sputum was strongly positive for tubercle bacilli.

Her married life was happy and she and her husband appeared devoted to one another, but there were no children and she had few interests apart from weekly visits to the cinema. Mentally she was anxious and worried and required a considerable amount of reassurance before she would settle to treatment. This reassurance was made more difficult by the fact that she was not a particularly intelligent subject, and it seemed impossible for her to grasp the fact that her co-operation was essential if a cure was to be achieved. In spite of prolonged rest her condition deteriorated and, collapse therapy having been refused, she was discharged at her own request in July, 1933, to continue treatment at home.

A year later she was readmitted in a much more serious condition with bilateral pulmonary disease and a laryngeal lesion. Her doctor reported that she had been very "nervous" lately and had been suffering from insomnia. She failed to settle to sanatorium régime, the insomnia grew

more troublesome and she became increasingly depressed. On October 14, 1934, she ran away from the sanatorium by way of a fire-escape and made her way home. The combined persuasive efforts of her husband and her doctor prevailed upon her to return a week later, but by that time she had begun to threaten suicide. A table-knife was found under her pillow one morning, while the same evening she confessed to her husband that she had made an attempt to hang herself with the electric-light cord. Two days later she again contrived to escape, in spite of close supervision, and the following week she was seen by a psychiatrist and admitted to a mental hospital, where the superintendent reported her as "suffering from a fairly severe depression with periods of extreme agitation each evening, when she imagined she was about to be imprisoned, tortured and put to death". Under these conditions the pulmonary disease went steadily ahead, and she died on July 31, 1935.

Here we have the case of a rather unintelligent woman at the menopausal age faced for the first time in a sheltered and protected existence with a crisis in the shape of a chronic and disabling disease. Anxiety was an early symptom, which repeated reassurance and explanation failed to allay; and as this anxiety increased, accompanied by agitation, she began to go steadily downhill, in spite of the fact that the initial pulmonary lesion was relatively small in extent. It is not suggested that the mental illness was the direct result of the tuberculosis, but the latter was obviously the precipitating cause, and the onset of the mental complication contributed largely to the fatal outcome.

**CASE 2.**—A married woman aged thirty-two was admitted to the sanatorium on December 3, 1934. She gave a history of pleurisy with effusion and haemoptysis in 1931, for which she was not treated and which, until directly questioned, she declared she had forgotten. Six months later she had married, and she remained reasonably well until June, 1934, since which time there had been a progressive deterioration in her health, and her husband, a medical practitioner, reported her as being "nervous and worried", so much so that he had persuaded her to return to her parents' home for a time.

On admission there was radiological evidence of infiltration in the upper and mid zones of the right lung. Sputum was T.B. positive and the blood sedimentation rate was 31 mm. (Westergren). She was mentally depressed and extremely introspective, and on questioning admitted that her main worry was the knowledge that she had tuberculosis and felt she would not recover. In spite of every effort being made to reassure and encourage her she failed to settle to treatment and mental depression persisted, though her general condition improved somewhat, temperature and pulse became normal and there was X-ray evidence of healing of the pulmonary lesion.

About four months after admission there was a sudden exacerbation of the mental symptoms and she was seen in consultation by a psychiatrist, who stated that he found her "lacking in concentration and initiative and

worried over these symptoms". He was of the opinion that there was a definite mental instability which was being aggravated by ill-health, and he considered that there was a suicidal tendency present.

For the following three months there appeared to be some improvement in her condition both mentally and physically, sputum had become T.B. negative and she began to get up for a short time daily.

This state of affairs did not endure, however. Mental depression returned in full force after a few weeks, and she volunteered the information on this occasion that she did not wish to recover, as she felt she was a burden on her husband. Following this recrudescence of the mental symptoms her physical condition began to deteriorate, the pulmonary disease extended and she died some two months later.

In this case the evidence again suggests a pre-existing mental instability which, however, was not evident until the patient was subjected to the stress and strain of prolonged illness. The chief factor in her psychological collapse was the fear of chronic disablement, and this fear persisted even during those periods when physically she was undoubtedly making headway. Why this fear should have been so persistent was never clearly established, but it is probable that the knowledge of the breakdown prior to her marriage (the significance of which she fully appreciated at the time but which she contrived to ignore in view of her marriage) may have been a determining factor, and that she felt the ostrich-like attitude which she had adopted at that time had led to the ruin of her life.

CASE 3.—A young woman aged twenty-nine, unmarried, was admitted on May 7, 1934. She had had sanatorium treatment elsewhere previously, and her former medical advisers reported that she was an extremely difficult patient. On admission she was under weight and mildly febrile; the blood sedimentation rate was 38 mm. in the first hour and sputum was T.B. positive. The skiagram showed infiltration involving the upper zones of both lungs, with a small cavity below the left clavicle. Prior to admission a left pneumothorax had been attempted but without success, and subsequently a left phrenic evulsion was carried out.

She was intensely introspective and took a delight in delving back into her medical history from infancy onwards, writing to a succession of doctors through whose hands she had passed and obtaining from them details of the minor illnesses of her childhood. For some weeks after her arrival it was impossible to enter her room without emerging laden with a fresh bundle of medical detail.

Although of an excitable nature she made a genuine attempt to settle to sanatorium treatment, and at first appeared to be making progress. In August, 1934, however, there was a brief febrile attack which necessitated a continuance of bed rest, during which time she occupied herself with the compilation of a family tree and researches into her family history. In the course of those researches she unearthed several facts which she considered discreditable, and this discovery had a disastrous effect upon her. Around

those facts, very minor ones in themselves, she built up an elaborate theory that she was now suffering in expiation of faults committed by her ancestors, and from that proceeded to manifest delusions concerning her own past, which included amongst others an assault committed upon her by a high church dignitary. For the remainder of her stay in the sanatorium she lived in a state of mental confusion, disorientated as to time and place, and there was a concomitant gradual deterioration in the pulmonary condition. Later she developed delusions of persecution and finally, as she was becoming violent and destructive, she was removed on the advice of a psychiatrist.

When last heard of in 1939 she was still alive, in spite of advanced pulmonary disease, but there had been no improvement in her mental state.

There is little doubt in this case that the mental instability had been present prior to the development of the pulmonary disease, and indeed it is probable that the rôle played by the latter was a very minor one. The mental symptoms suggest a schizophrenic type tending to probe closely into things, as evidence of which we have her enquiries into her own early medical history and her family history. While the influence of the pulmonary disease on the mental symptoms may be rather difficult to assess here, there is ample evidence that the latter had a most adverse effect on the lung condition and that from the appearance of the more obvious and definite mental symptoms her condition steadily deteriorated.

The next two cases come into a rather different category and are described as examples of an acute confusional insanity with recovery, arising in the course of pulmonary tuberculosis.

CASE 4.—A young naval officer aged twenty-seven was admitted on January 19, 1932, with a history of cough, sputum and fever of three and a half months' duration. He was running a temperature up to 100° each evening, sputum was T.B. positive and the X-ray showed infiltration and cavitation in the right upper zone and infiltration in the left mid zone. Shortly after admission there was an extension of the disease on the left side and a course of sanocrysine was begun. In April he was feeling better and temperature was settling, but a few weeks later he began to complain of aching in the shoulders, insomnia and general restlessness. These symptoms persisted for about a month, then the temperature rose sharply, the restlessness increased and was now accompanied by definite mental confusion and disorientation. An interesting point noted at this juncture was a rise in blood pressure, which read 190/150. The blood urea and urea concentration test were normal. Owing to the patient's extreme restlessness and continued efforts to get out of bed, he had to be kept under very close observation. The disorientation as to place was complete, and at one moment he was on board ship, the next in a front-line trench in France, while hallucinations of sight and hearing were frequent. His room was peopled with pipers and drummers marching round his bed, and on one

occasion he told us of a race between his bladder and a French bladder, which ran along neck and neck until the French bladder burst !

He was seen by a psychiatrist, who made a diagnosis of an infection-exhaustion psychosis and prescribed a course of bowel lavage, to which the patient reacted immediately. The temperature slowly fell, blood pressure became normal, he began to gain weight and thereafter made uninterrupted progress.

CASE 5.—A man aged fifty-one was admitted on November 25, 1938. He occupied an extremely important post in the business world and had recently achieved his life's ambition in being appointed general manager of his company. He was extremely ill on admission, with high fever, and X-ray examination showed the right lung to be densely consolidated throughout. The sputum was T.B. positive, and shortly after admission a right artificial pneumothorax was induced.

Early in January, 1939, he developed symptoms of mental confusion, with delusions. The latter were mainly concerned with his work, and he dealt in fancy with large sums of money and transacted much important business over a period of a few weeks. He remained quite cheerful through it all, although for a time he appeared distressed on account of a supposed offer of a knighthood, which for various reasons he did not desire to accept. At the end of February the mental condition gradually cleared up, and by the middle of March he was quite normal again.

During the succeeding two months temperature fell to normal, but in June he developed an effusion in the pneumothorax which persisted throughout the summer, finally becoming purulent. His general condition suffered and he began to lose ground, with the result that in January, 1940, the mental symptoms recurred. Again his delusions were largely concerned with his business, and for days he sat in bed addressing meetings of his board of directors, although his nights were frequently spent in a shed with sheep or in a cricket pavilion ! A feature of the condition at this time was that temperature remained only slightly raised and the blood sedimentation rate showed a decided fall. He began to refuse food and eventually declined even to take fluids by mouth, so that for a time it looked as though he was not going to recover. He was put on a continuous rectal drip, and finally a series of therapeutic "talks" were begun, which consisted of the frequent repetition of the same thing—namely, the imperative necessity of his pulling himself together if he wished to survive. On the first occasion there was little response, but on the second and third the gist of the message appeared to penetrate, and from that point improvement set in, and after about a week his mental condition was quite normal.

In these latter two cases the conclusion seems warranted that the pulmonary tuberculous disease had a very definite responsibility in the matter of the mental breakdown. Both patients were normal in their behaviour beforehand, both were highly intelligent, rational subjects, and both were intensely devoted to their work. Case 4 had the mortification of seeing his

service cut short in its early stages, while Case 5 had no sooner attained his ambition than the cup was dashed from his lips by this shattering physical breakdown. The toxic element undoubtedly played a part in both instances, but one feels that bitter disappointment and frustration may have turned the scales and they sought refuge in fantasy from the realities of the situation. With the subsidence of the toxæmia readjustment took place and they found themselves able to cope with the difficulties. One is tempted to speculate as to whether these symptoms were the result of a special variety of toxæmia in these cases, but no other focus beyond the pulmonary lesion was found in either individual, and one must conclude that the toxins emanated from this source.

#### Summary

Five cases of mental disturbance associated with pulmonary tuberculosis are described and an attempt made to elucidate the connection between the two conditions. In the first three it is concluded that a mental instability was present previously and that the tuberculosis was merely the means of bringing it to light and was in no way the direct cause; while in the remaining two cases the tuberculous toxæmia and their own special circumstances combined to produce an acute confusional insanity which cleared up with an improvement in their physical condition.

I wish to express my indebtedness to Dr. A. G. W. Thomson, Commissioner of the Board of Control for Scotland, for his helpful advice and criticism in the preparation of this paper.

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## COD-LIVER OIL AS LOCAL TREATMENT FOR TUBERCULOUS LESIONS

BY ANDREW L. BANYAI,

M.D.,

Muirdale Sanatorium, Wauwatosa, Wisconsin, U.S.A.

THE object of this paper is to present my experience with the topical application of cod-liver oil in 270 cases. I began the external use of cod-liver oil in a case of lupus vulgaris in October, 1934. A preliminary report on this patient, together with my observations on other types of tuberculosis, such as suppurating lymphadenitis, ischio-rectal fistula, empyema, subcutaneous abscess, fistula secondary to epididymitis, and so-called primary tuberculosis of the muscle, was published by me previously.<sup>1</sup> My results with the topical application of cod-liver oil in tuberculosis of the upper respiratory tract were reported in another paper.<sup>2</sup>

I received the encouragement for the local use of cod-liver oil from the works of Loehr,<sup>3</sup> who employed it in a large number of non-tuberculous conditions with success. He stated that cod-liver oil exerted a striking inhibitory effect upon the bacterial flora of wounds. He found also that bacteria ordinarily encountered in infected wounds, such as streptococci, staphylococci and *Bacillus coli*, perish when introduced into cod-liver oil.

The favourable results from the use of cod-liver oil and tomato juice in intestinal tuberculosis, a treatment first recommended by McConkey,<sup>4</sup> were seen frequently enough in my practice to suggest a search for some therapeutic factor in this combined medication. McConkey expressed the idea that this treatment effects some change in the calcium metabolism, which brings about an immediate relief of symptoms, and that the increased appetite and improved nutrition which follow the relief from intestinal symptoms allow the ulcers to heal. During the treatment of my patients the question arose whether the vitamin A in the cod-liver oil is responsible for the beneficial results rather than the vitamin D.

It seemed that there are two possible explanations for this assumption. First, that the vitamin A may come in direct contact with the ulcerated areas. It was realised that intestinal absorption prevents most of the ingested oil from reaching some of the distally located intestinal ulcers; on the other hand, considering that the peristalsis is often increased and the absorption is incomplete in intestinal tuberculosis, the possibility of a direct contact between oil and the ulcers could not be entirely disregarded. Secondly,

it was thought the absorbed cod-liver oil increases the vitamin A level of the blood and thereby induces healing of tuberculous ulcers of the bowels indirectly.

Previous bacteriological studies also supported the feasibility of the topical application of cod-liver oil. The possible bactericidal effect of animal oils upon the tubercle bacillus and their inhibitory effect upon its growth has been briefly summarised by Wells, DeWitt, and Long.<sup>5</sup> They quote the work of Miller, who stated in 1916 that the addition of sperm oil to media on which tubercle bacilli were grown caused rapid degenerative changes in the bacilli. Their own research work confirmed Miller's results. The investigations of Fontes are also quoted by them. Fontes found that the addition of 1 per cent. cod-liver oil, or chaulmoogra oil, or beef gall to ordinary broth culture medium prevented the development of cultures of tubercle bacilli. The oil in the fluid medium did not lose this inhibiting power when filtered through porcelain but did lose it when shaken with kaolin. Fontes thought that the inhibitory effect of cod-liver oil is due to the fact that tubercle bacilli absorb fat particles which form an isolating envelope and deprive the micro-organisms of nutrition and oxygen. Campbell and Kiefer<sup>6</sup> in 1922 demonstrated a definite inhibitory action of cod-liver oil upon the tubercle bacillus. Cotton-seed oil was used as a control, and it showed no inhibitory influence. They expressed the opinion that at least part of the inhibitory action was caused by some constituent of the oil and not altogether by a mechanical action of a film between the bacterium and the medium. The work of Campbell and Kiefer<sup>6</sup> justifies the inference that this constituent is vitamin A. They pointed out that cod-liver oil extracts made with a high degree of heat had much less inhibitory effect than extracts made at low temperature.

Platonov<sup>7</sup> investigated the effect of saturated and unsaturated soaps of cod-liver oil upon the growth of tubercle bacilli on potato media. The concentration of the soaps in the media varied from 0·25 to 4 per cent. After three weeks he found that unsaturated soaps, even in the concentration of 0·25 per cent., inhibited the growth of the bacilli, while the saturated soaps gave the same effect only in concentrations exceeding 4 per cent. When an emulsion of tubercle bacilli in a 1 or 2 per cent. solution of unsaturated soaps was used, the effect was still greater. After 48 hours in the incubator, the preparation stained by the Ziehl-Neelsen method had an interesting appearance: the tubercle bacilli were pale pink, and contained dark granules as though they were stained with methylene blue; furthermore, a considerable number of bacilli lost their acid-fastness.

A careful search of the *Cumulative Index Medicus* since 1926 failed to reveal any publication in the literature dealing with the topical application

of cod-liver oil in tuberculous pharyngitis and laryngitis. My observations in these conditions seem to be worthy of recording. The treatment consisted of spraying the throat with cod-liver oil by means of an ordinary atomiser three times a day. Ordinary medicinal cod-liver oil was used. Sterilisation of the oil is not necessary; it is free of bacteria. The following case reports are typical illustrations.

L. L. (No. 10,103), twenty years old, white, female. Diagnosis: (1) Far-advanced pulmonary tuberculosis; (2) tuberculosis of the tonsils and peritonsillar structures; (3) tuberculous laryngitis. The patient stated that her throat condition developed two months before her admission to the sanatorium. She complained of sore throat and dysphagia. Both anterior and posterior pillars and both tonsils were markedly congested, the right tonsil and the right anterior pillar were ulcerated. The same involvement was present on the left side, although not very extensive. The treatment consisted of cleansing the throat with Dobell's solution and spraying it with cod-liver oil three times a day. *In three weeks' time* the ulcers disappeared, the margins of the pillars were smooth, and the congestion of these structures cleared entirely, except the posterior part of the right tonsillar fossa; the latter was found normal four weeks later. The soreness of the throat and dysphagia disappeared.

F. A. (No. 10,326), thirty-seven years old, white, male. Diagnosis: (1) Far-advanced pulmonary tuberculosis; (2) tuberculosis of the pharynx. The patient complained of marked dysphagia and sore throat. Because of the pain in the throat he was using a cocaine spray several times a day prior to the beginning of the cod-liver oil treatment. There was an ulcer the size of a bean in the left tonsillar fossa. The ulceration extended to the posterior pillar. An ulcer of approximately the same size was visible on the posterior pharyngeal wall to the right of the uvula. The balance of the pharyngeal structures was moderately congested. After one month's treatment with cod-liver oil spray to the pharynx the ulcers healed, except a split-pea sized area of the left tonsillar fossa that still showed some membrane. At this time the treatment had to be discontinued because the patient developed a tuberculous meningitis that caused his death. Observations made during the course of treatment showed the gradual clearing of the ulcerated, necrosed areas and the disappearance of the precipitate that covered the ulcers.

In my third case of tuberculous pharyngitis, A. E. (No. 10,106), twenty-three years old, white, female, a tuberculous meningitis terminated the treatment also. At the time when the patient was first seen she complained of intense sore throat and dysphagia. The posterior pharyngeal wall, the uvula, and the anterior pillars showed extensive "moth-eaten" ulcerations.

The mid-section of the right anterior pillar revealed a marked tissue loss. The tonsils had a chewed-out appearance. All ulcerated and necrotic areas were covered with a white exudate. The entire extent of the throat was markedly congested. Multiple tuberculous ulcerations were present at the root of the tongue. This patient was in the preterminal stage at the time of her admission. Still, in spite of her low vitality, a clearing of and an improvement in the ulcerated areas were detectable during the first two weeks of treatment with cod-liver oil spray.

Complete healing followed the topical application of cod-liver oil in another case: B. S. (No. 9,381). Improvement was noted in one patient. The treatment was of too short duration in two cases, and it had to be discontinued in one because the patient complained of soreness following the spray. He died three days later.

Altogether 164 cases of laryngeal tuberculosis were treated by cod-liver oil spray at Muirdale Sanatorium. Of these, however, only 91 were analysed as to the therapeutic effect; the balance represents those whose treatment lasted for less than two months. Active pulmonary tuberculosis was present in all patients. The laryngeal findings were those characteristic of tuberculosis associated with a chronic pulmonary process. Several patients had the combination of the different manifestations of the disease, such as infiltration, ulcerations, interarytenoid vegetative granulation, and oedema.

In patients who responded favourably to the treatment the relief from disagreeable symptoms was rather prompt. In several of the ulcer cases, who used cocaine spray, anaesthesia or euphagine tablets before the treatment, we were able to discontinue these shortly after the use of cod-liver oil began. The disappearance of dryness, tickling, and burning sensation from the throat, the cessation of pain that was localised to the larynx or radiated to the ear were frequently observed. With the elimination of the pain, dysphagia was relieved and the patient's nutrition and general well-being improved. It was also noted that the cough diminished and expectoration became easier. With the diminished cough and lessened expectoration, more pulmonary and general rest and a better, more quiet sleep were ensured for the patient. The gradual disappearance of hoarseness and the restoration of normal voice were very impressive for the patients as well as for the attending staff. Such favourable changes were followed by an improved, more optimistic mental attitude on the part of the patient.

The change in the laryngeal findings was striking as the treatment progressed. Ulcers showed a rapid epithelialisation and healing that was comparable with the results seen in pharyngeal cases, provided the treatment was carried out consistently and well tolerated by the patient. Similarly, a good therapeutic response was observed in patients with various degrees

of vegetative granulation. Other manifestations of the disease cleared up also satisfactorily. We had the least success in cases with marked oedema. Utter failure of the treatment in several of these cases was discouraging.

The analysis of the results in 91 patients in whom the treatment was continued from two months to one and a half years revealed that 24, or 26·3 per cent., remained unimproved (of these, 8 died during the course of treatment); 42, or 46·2 per cent., improved subjectively and objectively; 25, or 27·5 per cent., healed.

Two patients with tuberculous ulceration of the tongue were successfully treated by the topical application of cod-liver oil. At first an attempt was made to aid healing by applying the oil with an applicator at frequent intervals. At the suggestion of one of my associates (Dr. J. P. Schlaikowski) this procedure was substituted by having the patient hold small amounts of oil in the mouth and bathe the ulcerated area of the tongue in the oil by lying on the corresponding side.

The injection of cod-liver oil into the pleural cavity was first reported by Bezançon and his associates.<sup>8</sup> They attempted to influence the course of pulmonary tuberculosis in this fashion. The impression was gained on a small group of patients that the treatment was of benefit and the lesions tended to become stabilised. The explanation of this effect was that the collecting lymphatics of the visceral pleura, which are the same as for the superficial parts of the lung, communicate with the deeper pulmonary vessels which follow bronchi, arteries, and veins toward the hilum. *Severe reactions, however, seemed to render this procedure impracticable in patients who have no pleural effusion.* These reactions consist of sharp pain on the side of the injection, shallow respiration, nausea, pallor, sweats, and weak pulse. By the third day a pleural exudate develops and is always accompanied by fever. Reactions were not encountered in patients with a pleural exudate. For the injection either pure cod-liver oil was used or it was diluted with olive oil to from 10 to 50 per cent. The treatment was designated as oleomorrhuo thorax.

We have treated seventeen cases of empyema secondary to pulmonary tuberculosis. If there is no bronchopleural fistula present, a tuberculous empyema responds favourably to aspiration of the pus and to its replacement by small amounts of cod-liver oil. Patients in whom the empyema was evacuated by closed drainage or by costectomy were treated by the injection of 45 to 300 c.c. of cod-liver oil into the thoracic cavity. None of these patients improved on previous appropriate measures. In the absence of bronchopleural fistula the treatment was well tolerated. No local or general reactions were observed. In two patients the purulent pleural exudate was reduced to a minimum and thus they became eligible for

thoracoplasty for the purpose of obliterating the infected, gaping pleural cavity. I have not observed the gradual symphysis of the pleuræ as the result of oil injections. The explanation of this may be the marked thickening and rigidity of the visceral pleura. If a marked thickening and rigidity of the visceral pleura are absent, cod-liver injections can be used for the production of pleural adhesion and obliteration of the pleural space in artificial pneumothorax, as reported by Blanco Vitorero in 1936.<sup>9</sup> In the presence of bronchopleural fistula the amount of injected oil should be regulated according to the level of the pleural opening. At the time of injection it should be kept below this level to avoid a sudden coughing spell. The treatments had to be discontinued in one case because of such reactions, and in another case because the injection of even small amounts of oil was followed by a severe dyspnoea and cyanosis. The latter patient had a similar reaction after the intrapleural injection of gentian violet. By the effect of the oil upon the secondary pyogenic micro-organisms and by its influence upon the diseased pleuræ it was possible to reduce the pus formation substantially. In no instance have we seen the healing of the bronchopleural fistula. Failures of this treatment were encountered in patients with marked general debility or amyloidosis.

Satisfactory results were found in four patients in whom the oil was applied externally following thoracoplasty when the operation wound showed a delayed healing tendency. It was noted that cod-liver oil aided the formation of healthy granulation, reduced purulent secretion, and accelerated epithelialisation. In two instances in which an infection with *B. pyocyaneus* occurred the treatment was without any effect.

We treated five patients with post-operative abdominal fistulas that followed laparotomy for tuberculous peritonitis or presumably for an acute appendicitis. In the latter group tuberculosis of the cæcum was the cause of the fistula. The injections of cod-liver oil into the fistulous tract were preceded by irrigations with green soap, hydrogen peroxide and saline solution. In one case treatments were stopped because of lack of tolerance. No improvement was noticed in a case of post-operative ileo-cæcal fistula. In the other patients gradual improvement and closure were accomplished.

Good results were observed in eight cases of tuberculous lymphadenitis. One case not previously reported is given in detail—R. D. (No. 10,941), twenty-three years old, white, male. Diagnosis: Moderately advanced pulmonary tuberculosis. During his sanatorium stay he developed a tuberculous adenitis of the right cervical lymph nodes. A fluctuating mass, the size of a small apple, was aspirated on three subsequent occasions, and the pus was replaced by cod-liver oil. The amount of oil was about one-fourth of the pus removed. Complete healing resulted.

If the overlying skin is very thin, rupture of the abscess cannot be prevented by cod-liver oil injection. It must be emphasised, however, that the topical application of cod-liver oil is of great value in the treatment of broken-down abscesses that originate from a tuberculous adenitis. I have seen recovery in a rather surprisingly short time in cases that resisted prolonged heliotherapy and deep X-ray therapy.

The purpose of the treatment in such cases is twofold. First, the possible prevention of the invasion of pyogenic micro-organisms, and, secondly, the saturation of the involved tissues with oil and thus the promotion of granulation and epithelialisation.

We treated six patients with tuberculous phlyctenular conjunctivitis and kerato-conjunctivitis. It is realised that the etiology of phlyctenular conjunctivitis is considered an unsolved problem by many. Others, when it occurs in an individual with some type of active tuberculosis, consider it a sign of invasion of tubercle bacilli into the bloodstream, an indication of a possible haemogenous spread of the disease, or as an indication of an allergic response of the sensitised conjunctiva. Furthermore, it is also realised that phlyctenular conjunctivitis is usually benign, or it responds favourably to local heat application and to ointments, such as yellow oxide of mercury. My limited experience shows that cod-liver oil eye drops instilled into the conjunctival sac three times a day produce better and prompter response than other measures. Stevenson<sup>10</sup> has written on cod-liver oil as local treatment for external affections of the eye. He found in 150 patients that cod-liver oil seems to promote the growth of natural tissues and to inhibit the growth of the scar tissue in any case in which there is loss of substance. Rinaldi<sup>11</sup> treated twenty patients with corneal disease, including dystrophic, disciform, and phlyctenular keratitis, locally with vitamins A and D. In seventeen cases rapid healing followed the institution of treatment.

One of my cases, W. F. (No. 9,384), is described briefly. This patient, a fifteen-year-old coloured boy, was a typical example of a generalised haemogenous tuberculosis. Besides his pulmonary process, he had multiple bone tuberculosis, tuberculous lymphadenitis and multiple widespread tuberculous subcutaneous abscesses. He developed a phlyctenular kerato-conjunctivitis. Cod-liver oil treatment was given for five days. This was discontinued at the suggestion of the visiting ophthalmologist, who recommended the use of a 2 per cent. dionin solution three times daily. During the subsequent two days the condition of the eye got worse, whereupon the dionin treatment was discontinued, and cod-liver oil instillations were started again. Complete healing was accomplished by the subsequent four days' treatment.

In two cases of tuberculous keratitis cod-liver oil failed to produce satisfactory results.

This treatment was used in eighteen cases of bone tuberculosis. It does not need special emphasis here that the treatment of cold abscess must be focused primarily upon its source, the tuberculous process in the bone. It has been my practice to follow the institution of appropriate measures for the immobilisation of the osseous lesion by repeated removal of the pus by aspiration. After each aspiration cod-liver oil is injected. The amount should be about one-fourth of the pus evacuated. In this manner we were able to prevent sinus formation in some of our cases. It was noted that the oil increases and accelerates the liquefaction of the contents of a cold abscess. Thus it renders subsequent aspirations easier. Injection of too much oil may cause a sudden breakdown of the surrounding tissues and may lead to sinus formation. No pain or discomfort was observed following cod-liver oil injections. Parallel with the improvement in the bone focus, this treatment is likely to induce a subsidence of a cold abscess, provided treatment is started prior to an imminent sinus formation.

All familiar with the course of a broken-down cold abscess and fistulous bone tuberculosis realise the difficulties connected with their treatment. Besides proper attention to the original tuberculous process, we applied cod-liver oil dressings to the sinus, or injected the oil into the sinus, or both. The purpose of this treatment was twofold. First, to influence the lesion of the soft tissues, and, secondly, to protect the sinus from the invasion of secondary pyogenic micro-organisms. It is gratifying to see how readily some of these tuberculous fistulas respond to the topical application of cod-liver oil.

In instances in which the underlying bone lesion was very extensive and destructive such treatment was of no value whatsoever.

Seven patients were treated for tuberculosis of the genito-urinary system. Fistulas secondary to a tuberculous epididymitis responded favourably to injections into the fistulous tract and to cod-liver oil dressings in three cases, one of whom had renal tuberculosis also. In one patient satisfactory improvement of a post-nephrectomy sinus was noted. Two patients in whom the diagnosis was renal tuberculosis were treated by daily instillations of cod-liver oil into the bladder. Both complained of frequency and dysuria before the treatment. The cystoscopic examination showed congestion of the bladder mucosa but no tubercles or ulcers. The instillations of one ounce of oil were well tolerated by the patients. Because no substantial change was observed in the subjective feeling of the patients or in the cystoscopic findings the treatments were discontinued after three months and eight months respectively. Relief from dysuria and partial relief

from haematuria were seen in a patient, W. C. (No. 10,582), thirty years old, white, male, who, subsequent to a nephrectomy for renal tuberculosis, was admitted to the sanatorium with the diagnosis of tuberculosis of the urinary bladder. The length of treatment was two months. The patient left the institution against medical advice. Improvement of urinary frequency was observed in a patient with tuberculous ulcers of the bladder. The post-mortem examination of this patient, J. B. (No. 9,156), thirty-seven years old, white, male, revealed a complete destruction of one kidney by tuberculosis, the formation of a perinephritic abscess extending to the bony pelvis, and an unusually marked ulceration of the bladder. There was also evidence of an extensive pulmonary tuberculosis.

Nineteen tuberculous ischio-rectal fistulas were treated by daily injections of cod-liver oil into the fistulous tract. The treatments were given with the patient in the prone position. After each injection the patient was kept in this position for about twenty minutes. There was only one case in whom the treatment had to be discontinued because of pain caused by the injected oil. Satisfactory results were observed in the great majority of cases in whom the injections were continued for two months or longer. In some instances it was necessary to incise the external opening of the fistula to render the fistulous tract more readily accessible for the oil.

We treated by the topical application of cod-liver oil four cases of scrofuloderma, five cases of lupus vulgaris, and one case of multiple tuberculous subcutaneous abscesses. The results were invariably good in scrofuloderma. Of the five cases of lupus vulgaris, three made a complete recovery in three, four, and eight months respectively. One patient, N. Z. (No. 8,348), thirty-three years old, white, female, who had been suffering from skin tuberculosis for twenty years, and whose lesion involved practically the entire extent of the skin of the left arm, improved to a certain extent but has not healed completely in spite of a prolonged local treatment. Another patient, A. P. (No. 5,883), twenty-eight years old, white, female, whose lupus was of nine years' duration, showed rather prompt and rapid response to cod-liver oil. During the past four months the process, that extended over the sternum from the manubrium to the xiphoid process, healed with the exception of a pinhead-sized area at the top and at the bottom of the lesion. Subcutaneous abscesses, from which tubercle bacilli were isolated, in a case of generalised haematogenous tuberculosis, W. F. (No. 3,984), responded favourably to treatment by aspiration and cod-liver oil injections.

#### Conclusions

1. A report on the topical application of cod-liver oil in 270 cases of tuberculosis is presented.

2. Cod-liver oil can be used with safety in the conditions described above.

3. Pharyngeal and laryngeal ulcers showed a rapid epithelialisation and healing. Favourable therapeutic response was seen in laryngeal tuberculosis with localised infiltration and with a vegetative granulation. Laryngeal tuberculosis with marked oedema is rather resistant to this treatment.

4. Tuberculous empyema in the absence of bronchopleural fistula responds favourably to aspiration of the pus and to its replacement by cod-liver oil. The presence of a bronchopleural fistula does not contraindicate the use of the oil, but the results are much less satisfactory.

5. Suppurating tuberculous lymph nodes, when seen early, heal well on repeated aspirations followed by injections of cod-liver oil.

6. The oil when used in the form of eye drops is harmless. It induced more rapid recoveries in our phlyctenular conjunctivitis and keratoconjunctivitis cases than other methods of treatment. The results were unsatisfactory in tuberculous keratitis.

7. Cod-liver oil is of some value in treating cold abscess secondary to bone tuberculosis, provided proper attention is paid to the treatment of their source.

8. Fistulas originating from tuberculous epididymitis healed rather promptly on cod-liver oil injections. The response in tuberculosis of the urinary bladder was disappointing, although there was some improvement in frequency and dysuria.

9. Satisfactory results were observed in the great majority of ischio-rectal fistulas.

10. The results were invariably good in scrofuloderma. Four out of five cases of lupus vulgaris responded favourably to the topical application of cod-liver oil.

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## NATURALLY OCCURRING TUBERCULOSIS IN DOGS AND SOME OTHER SPECIES OF ANIMALS

By R. LOVELL AND E. G. WHITE,  
Research Institute in Animal Pathology, Royal Veterinary College.

### I. TUBERCULOSIS IN DOGS

#### Introduction

Dogs may be infected under natural conditions with tubercle bacilli of the human and bovine types and figures giving the relative incidence are available. The pathogenesis of tuberculosis in the dog has also been studied, but there are only scanty data of any correlation between the type of infecting bacillus and the character and distribution of the lesions produced. An opportunity occurred to make such a combined study on dogs examined post-mortem at the Royal Veterinary College.

During this period of study a few cases of tuberculosis were encountered in other species, and the data relating to these will be given in the second part of this paper.

#### Material and Technique

With the exception of one case (No. 4) all the dogs were brought to the Clinics of the Royal Veterinary College and Hospital and destroyed for various reasons.

A routine autopsy was made as soon after death as possible, and material for bacteriological examination was transferred to sterile containers. Smears were made and tissue for histological examination was fixed in formol saline. Sections were usually cut by the freezing method on the day after the autopsy, and also after embedding the material in paraffin: they were stained by Ehrlich's acid haematoxylin and eosin and, in the case of paraffin sections, by Ziehl-Neelsen's method. It was often difficult to find acid-fast organisms in smears made from the lesions and, in some cases, the examination of frozen sections gave the first definite evidence of tuberculosis, although in every case the diagnosis was confirmed by the isolation of tubercle bacilli.

Cultures were obtained either by direct methods or from guinea-pigs inoculated with original material. The cultural characteristics of the strains were tested by growth on egg medium and on inspissated serum, with and without the addition of 5 per cent. glycerin. Cultures were incubated at 37° C. and examined at intervals over a period of three months. Except

where otherwise stated, virulence tests were made by the intramuscular injection of young cultures into guinea-pigs and rabbits. Animals which were not dead at the end of four months were killed—all animals were examined post-mortem. The dose employed is expressed in milligrammes of moist culture, although estimations were made by opacity tests, using as standard a killed twenty-four-hour culture of *Bact. coli* containing 1,000 million organisms per c.c. The error involved in the estimation of the dose is great, but it is clear from the work of Wilson and Schwabacher (1937) that the method here employed is of greater accuracy than many: these authors consider that the number of tubercle bacilli contained in one milligramme of moist growth is of the order of 1,000 to 1,500 million.

### Results

During the years 1938-39 we examined 543 dogs post-mortem, and of these 25 were tuberculous—a percentage of 4·6. Of these, 22 were examined bacteriologically, and tubercle bacilli were isolated from thirteen by direct means and from the remainder by guinea-pig inoculation. The human type was isolated from 18, three strains being of moderate or slightly reduced virulence; the bovine type was recovered from the other four dogs.

The detailed protocols are given at the end of the paper in the form of an appendix.

### Discussion

*General Incidence.*—The recorded incidence of tuberculosis in dogs varies according to whether it is based on clinical or autopsy data, being usually higher with the latter. A selection of published data is given, and it will be seen that it varies from 0·04 per cent. to 1·0 per cent. for clinical statistics and from 0·83 per cent. to 5·6 per cent. for autopsy material. Our percentage based on autopsy data is 4·6. It must be noted, however, that the dogs brought to the clinics of the Royal Veterinary College Hospital are not a random sample; they are largely the property of poor people living in London.

*Sex.*—In our 22 cases of tuberculosis there were 19 male dogs and 3 bitches; the ratio of males to females among dogs coming to autopsy during the same time was 3 : 1. A higher incidence in males is also shown in figures given by Yost (1921), who records 75 cases of tuberculosis in 4,683 dogs examined. Of these 52 were males and 23 bitches; the sex ratio among the total of dogs examined was 1,724 males to 2,959 females.

*Breed.*—It is to be expected that a variation in the incidence according to the breed of dog might be correlated with the opportunity of acquiring infection, but our numbers are too small to show this. There were, however, in the 22 cases described here 8 wire-haired fox terriers (36 per cent.); of

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350 consecutive dogs received for autopsy the proportion of this breed was 11 per cent. Hjärre (1939) examined 175 cases of tuberculosis in Stockholm between 1908 and 1937, and the highest incidence was observed in Irish setters (10·6 per cent. of a total of 234) and fox terriers (10 per cent. of 379).

*Age.*—The average age of dogs affected with tuberculosis was, in our series, five years, and ranged from six months to twelve years. Hjärre (1939) states that tuberculosis is most common between one and five years and relatively rare in old dogs; in his large number of cases the age ranged from five months to thirteen years.

TABLE I.—INCIDENCE OF TUBERCULOSIS IN DOGS. (FROM FAULENBORG AND PLUM [1935].)

Author.	Year.	Town.	Number of Dogs.	Percentage Tuberculous.
<i>(a) Clinical Statistics :</i>				
Fröhner	1886-95	Berlin	62,500	0·04
Cadiot	1891-93	Alfort	9,000	0·44
Sendrail, Lassert and Lesbouyries	1913	Toulouse	1,430	0·8
Wirth	1922	Vienna	6,700	1·0
Hinz and Schröder	1925-26	Berlin	23,000	0·3
Mágies	1926	Budapest	—	0·2 (94 cases)
Smythe	1929	London	—	0·3-4·0 (20 cases)
<i>(b) Autopsy Statistics :</i>				
Eber	1893	Dresden	400	2·75
Petit	1900-04	Paris	2,717	5·6
Joest	1909	Dresden	2,156	0·83
Crocker	1919	Pennsylvania	1,548	1·0
Hjärre and Herlitz	1929-33	Stockholm	1,129	0·6-9·4
Nieberle and Pallaske	1933	Leipzig	630	5·2
The Authors	1938-39	London	543	4·6

*Clinical History.*—In one animal (No. 2) there was no ill-health apart from thickening of the spermatic cord. In three dogs (Nos. 3, 5 and 18) the symptoms consisted mainly of swelling of the mandibular lymph node, and in two of the dogs there was sinus formation and a small amount of purulent discharge. In the cases in which the mesenteric nodes were primarily involved the symptoms were usually vomiting and diarrhoea of a few weeks' duration. Those cases in which the lungs were solely or principally concerned were frequently admitted with a history of loss of condition and coughing which had been noticed for as long as one year or occasionally for as short a time as a few days. The temperature was in most cases 103° to 104° F. When both the alimentary and respiratory systems were involved there was a combination of symptoms—e.g., Nos. 17, 19 and 20.

The subcutaneous tuberculin test was applied in 8 animals, and gave a positive reaction in 5; in the remaining 3 cases the high pre-inoculation temperature rendered the interpretation of the reaction doubtful.

*Pathogenesis.*—Most workers consider that healed tuberculous lesions are rarely found in the dog, and this is our experience, for we have failed to find such lesions. On the other hand, Blair (1916) states: "Healed tuberculous lesions have been frequently observed in the lungs of dogs." In the series of cases recorded, tuberculous lesions were confined to a single organ or lymph node in only two animals, Nos. 4 and 6, and in the former the examination was incomplete. Feldman (1934) described a case of natural tuberculosis in the dog in which the disease was localised to the apical lobe of the left lung and was similar to one described in this paper (No. 6).

Nieberle gives the impression that, in the dog as in cattle, the primary lesion is easily recognised. In a total of 33 cases (Nieberle, 1932) he found primary lesions in the respiratory tract in 28 dogs; in 16 the primary lesions were present both in the lungs and bronchial nodes, and in the remaining 12 the bronchial nodes alone showed macroscopic lesions. Of the 5 cases in which the primary lesion was situated in the alimentary tract, one was in the throat and the others in the mesenteric lymph nodes. Hjärre (1939), in his study of 175 cases of canine tuberculosis, states that it is often difficult to determine with certainty the site of the primary lesion. In 162 of the animals he was able to identify its probable site; it was situated in the respiratory tract in 59 per cent., in the alimentary tract in 37 per cent., and was cutaneous in 4 per cent. In the last-mentioned situation there was usually the history of a bite. His studies indicate that tuberculosis caused by the bovine type more often shows a primary lesion in the alimentary tract, whereas most of the cases caused by the human type are ærogenous in origin. This author also states that he could find no difference between the tissue reaction in the dog to infection with the two types of tubercle bacilli. Our findings are in agreement with his. In all four of our 22 cases which were due to bovine tubercle bacilli the mesenteric nodes were involved; in Nos. 2, 4 and 8 the mesenteric or gastric nodes were the primary site and in No. 20 the primary lesion was probably in the alimentary tract. There were seven animals in which the primary lesion was situated in the respiratory tract only—Nos. 6, 7, 10, 12, 15, 21 and 22—and from all of these the human type of bacillus was isolated. Of the remaining cases, all due to the human type, Nos. 3, 5, 11, 13, and 14 were probably alimentary in origin, whilst Nos. 1, 9, 16, 17, 18 and 19 might have been either respiratory or alimentary or, possibly, represented primary infection in both sites. Whilst the number of cases examined was too small to justify an unreserved

opinion, there was no evidence that the tissue reaction differed macroscopically or histologically with the type of infecting bacillus.

*Lesions.*—Involvement of the serous membranes is common in canine tuberculosis. Of the 22 cases there was a tuberculous pleuritis in 9, accompanied in 6 cases by effusion; there was, in addition, one case of hydrothorax without pleural lesions. In 5 cases showing pleural lesions the omentum or parietal peritoneum was also involved. In all, there were 7 cases of tuberculous peritonitis, only one being accompanied by effusion, and in 5 of the 7 there were omental lesions. The latter were also observed in 3 additional cases. In all cases of peritoneal lesions except dog No. 22 there was tuberculosis of the mesenteric or portal lymph nodes or both. The serous covering of the diaphragm was involved in 10 animals, and was associated in every case with lesions on other areas of the parietal peritoneum or pleura. The portal lymph nodes were affected in 6 animals, and in 3 of these the mesenteric were also involved. The mesenteric nodes showed tuberculous lesions in 8 animals.

We have seen no record of primary tuberculosis of the tonsil in the dog, primary lesions in the throat being usually limited to the mandibular lymph node, at least as far as macroscopic examination is concerned. The condition described in dog No. 5 is therefore of interest. We have not observed primary tuberculosis in the stomach or intestines, and this is in agreement with the findings of others who record an incomplete primary lesion with lymph node involvement only in these sites.

It is generally agreed that calcification is extremely rare in tuberculosis in the dog. Hjärre (1939) has, however, recorded the presence of basophil deposits, probably calcium, and this has been our experience in dogs Nos. 1 (lung), 10 (liver), 18 (mediastinal node) and 22 (all lesions).

Giant cells are extremely rare in canine tuberculosis, and we have not seen any of the Langhans type, although they have been recorded by Mihailescu (1938).

*Bacteriology.*—There is a great variation in the number of tubercle bacilli found in smears and sections of tuberculous lesions in dogs. In our experience they have usually been scarce. The notable exception has been tuberculous pyelonephritis, where they were extremely numerous. In some cases it was necessary to search several smears before finding a single acid-fast organism. Stableforth (1929) found the bacilli scarce except in lung lesions, and stated that, in lymph-node lesions, long search might fail to reveal any. In the case recorded by Wilson and Lovell (1928) there were large numbers of bacilli. In 11 cases reported by Schornagel (1914) bacilli were numerous or very numerous in 6, scarce in 4 and absent in 1. Hebrant, Antoine and Stappers (1914) were unable to find acid-fast

organisms in smears in 2 of 5 cases, although both were positive on guinea-pig inoculation. The difficulty in finding tubercle bacilli in smears and the low virulence of the organism were responsible for the report of a case of canine tuberculosis by Baumann (1937) under the title "Mycosis fungoides".

The relative incidence of human and bovine type infections is comparable in our series with those of other workers. One record only of infection with the avian type has been found (Plum, 1936). Approximately three-quarters of natural cases of tuberculosis in dogs are caused by the human type of bacillus, the remainder being due to the bovine type (see Table II.). The association of tuberculous human beings with the disease in the dog and the high proportion of canine cases caused by the human type appears to be due more to the opportunity of infection than to any inherent difference of susceptibility of the dog to the two types (Cobbett, 1922). Occasional cases of tuberculosis due to the bovine type in dogs which frequent abattoirs are recorded by Nain and Prudhomme (1938).

TABLE II—TYPE OF TUBERCLE BACILLUS ISOLATED FROM NATURAL CASES OF TUBERCULOSIS IN DOGS.

Country.	Author.	Type.			Total No. Ex- amined.
		Human.	Bovine.	Avian.	
England	Griffith, 1928	1	—	—	1
"	Stableforth, 1929	10	6	—	16
Scotland	Wilson and Lovell 1928	—	1	—	1
U.S.A.	Smith, 1905	—	—	—	1
"	Feldman, 1930	1	—	—	1
"	Feldman, 1934	—	1	—	1
Germany	Zwick, 1908	1	1	—	2
"	Joest, 1909	—	1	—	1
"	Rabinowitsch-Kempner, 1921	16	1	—	17
France	Baumann, 1937	1	—	—	1
"	Panisset and Verge, 1924	2	—	—	2
"	Nain and Prudhomme, 1938	—	1	—	1
Holland	Schornagel, 1914	6	2	—	8
Hungary	Mihaleescu, 1929	8	—	—	8
"	Mihaleescu <i>et al.</i> , 1938	12	—	—	12
Denmark	Faulenborg and Plum, 1936	2	1	—	3
Italy	Plum, 1936	2	—	1	3
Dutch E. Ind.	Cirenei, 1938	—	1	—	1
Norway	Van Zweiten, 1938	2	—	—	2
England	Hjärre, 1939	51	23	—	74
	Here recorded	18	4	—	22

*Relation to Tuberculosis in Man.*—Of the 22 tuberculous dogs reported in this paper there were 3 in which a definite history of tuberculosis in the owner or other member of the family was obtained. Smythe (1929) recorded a history of tuberculosis in members of the family in 4 of 20 cases of tuberculosis in dogs. Stableforth (1929) was able to obtain an adequate medical history of the household in 6 of 16 cases, and in 4 of these there was a history of human tuberculosis. In Stockholm, Hjärre and Herlitz (1935) examined the history of the household in 25 cases of canine tuberculosis and elicited a history of tuberculosis in 6. Another opportunity for acquiring infection with tubercle bacilli of the human type occurs in dog, owned by proprietors of restaurants and public-houses. For examples Lesbouyries (1926) records that 8 of every 10 tuberculous dogs in Paris and its suburbs live in cafés or restaurants and the remainder in close contact with tuberculous human beings. He quotes Cadiot (1893), who found that 9 of 40 tuberculous dogs examined by him belonged to proprietors of restaurants or public-houses.

A detailed consideration of the cases recorded by Hjärre and Herlitz failed to provide evidence of transmission of the disease from dog to man, but the possible danger to human beings is stressed, the main sources of infection from tuberculous dogs being the sputum and urine. Cutaneous tuberculosis would also be a danger to man, although most cases described are in fact tuberculous adenitis of the mandibular or other superficial lymph node with secondary sinus formation. Liegeois and Bertran (1929), however, describe two cases of true cutaneous tuberculosis in the dog and suggest that infection may have occurred from the urine. Lesbouyries (1926), in his monograph on tuberculosis in domestic carnivora, states that tubercle bacilli are frequently found in the urine. Robin, Brion and Cosson (1934) inoculated guinea-pigs with urine from 26 dogs diagnosed clinically as tuberculous and obtained positive results for tubercle bacilli in 14. Nineteen of the dogs were finally examined post mortem and secondary renal tuberculosis was found in 7. That the bacilluria may be continuous over a period of time was shown by seven positive samples obtained from a dog during the course of a month. The authors state that a tubercle bacilluria can also exist in the absence of macroscopic renal lesions. On the other hand Mihailescu (1938) states that tubercle bacilli are present in the urine only when there is tuberculous pyelonephritis, which condition he considers rare in the dog. In our series there were two cases of tuberculous pyelitis, and in both tubercle bacilli were found in smears and sections in enormous numbers, both in the renal tissue and in the fluid within the renal pelvis; such cases must clearly result in a heavy infection of the urine. In addition, there were secondary

cortical and medullary foci in ten animals, but no urine examinations were carried out.

### Summary

A study of 22 naturally occurring cases of tuberculosis in dogs is recorded and the pathology and bacteriology discussed. From 18 of the dogs the tubercle bacilli were of the human type, the remaining 4 being bovine.

We are indebted to Mr. F. W. Withers, M.A., M.R.C.V.S., for making autopsies on some of the dogs.

### APPENDIX

No. 1.—Thirteen-months terrier, male; three months' dyspnoea, gradual loss of flesh and chronic cough; reacted to subcutaneous tuberculin test.

*Autopsy*.—Emaciated; pleural effusion; mass of mediastinal tissue (200 gm.) adherent to lungs, pericardium and diaphragm; partially collapsed lungs (140 gm.); plaques on parietal pleura; lungs studded with minute translucent, greyish foci; tracheo-bronchial nodes enlarged, firm, anthracotic. Liver studded with small greyish foci. Greyish lesion (5 mm.) in right ventricle. Mesenteric nodes slightly enlarged, two minute greyish foci on section. Few acid-fast bacilli in smears from liver lesions.

*Histology*.—Bronchial nodes showed no normal tissues; necrosis, hyalinisation, epithelioid cell tubercles at periphery. Two cortical lesions in right adrenal, one with central necrosis. Kidneys showed chronic nephritis, mainly involving glomeruli; dystrophic calcification of bloodvessels and degeneration of tubule epithelium; no tuberculosis. Submiliary tubercles both lungs, also hyaline necrotic areas with central basophil deposit, probably early calcification. Recent epithelioid cell tubercles in mesenteric nodes. No acid-fast bacilli found in sections.

*Bacteriology*.—Culture direct from marrow of femur, lung, mediastinal mass and liver. Good growth on egg; more luxuriant on glycerin media. Two guinea-pigs inoculated with original material died after 42 and 56 days respectively with generalised tuberculosis. Two rabbits receiving 10 mgm. of culture were killed after 56 and 210 days respectively: the former showed tuberculous local lesion and lymph nodes only, the latter was free from tuberculosis.

Eugonic human type.

No. 2.—Nine-years fox terrier, male. Thickening of left spermatic cord noticed three weeks prior to admission to clinic. Unilateral orchidectomy. Left testicle normal, spermatic cord irregularly thickened and adherent to tunica vaginalis. Frozen sections showed tuberculosis; epithelioid cells and numerous neutrophil leucocytes, a few plasma cells and lymphocytes. No giant cells, necrosis or calcification. Two acid-fast bacilli found in frozen sections. Tuberculin test positive. Animal, apparently in perfect health, destroyed one week after operation.

*Autopsy*.—Productive tuberculous pleurisy (plaques), a few greyish foci in liver; plaques on peritoneal surface of diaphragm; portal and gastric nodes enlarged, firm, greyish white on section; slight enlargement of right mandibular and left internal iliac nodes.

*Histology*.—None of the tuberculous lesions showed necrosis; they were composed of epithelioid cells, plasma cells and lymphocytes with large numbers of neutrophils. The latter feature is unusual in dogs except in lesions secondarily infected—e.g., tuberculosis of the mandibular lymph nodes with sinus formation. No tubercle bacilli found in sections.

*Bacteriology*.—Culture from guinea-pig inoculated with emulsion of sublumbar nodes of guinea-pig injected with original material from portal and gastric nodes of dog. Growth on egg and serum slow and poor; less growth on glycerin media. Two guinea-pigs inoculated with 0.1 mgm. of third generation culture died after 46 days with generalised

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tuberculosis. Two rabbits injected with 1.0 mgm. died after 59 and 78 days respectively with generalised tuberculosis.

Dysgonic bovine type.

No. 3.—Two-years fox terrier, male. Discharging sinus in neck in region of left mandibular node. No acid-fast organisms found in smears of pus, but cellular picture suggested tuberculosis. Tuberculin test positive. Animal destroyed.

*Autopsy*.—Sinus originated in enlarged left mandibular node; prescapular node on same side enlarged and firm. Corresponding nodes on right side slightly enlarged. Mesenteric and mediastinal nodes enlarged. Multiple greyish lesions throughout liver. Single plaque on pleural surface of diaphragm.

*Histology*.—All affected nodes showed tuberculosis with irregular areas of necrosis. No giant cells or calcification and very few neutrophils. No acid-fast organisms found in sections.

*Bacteriology*.—Culture from guinea-pig inoculated with emulsion of left mandibular node. Heavy confluent growth on egg and serum; improved luxuriant growth on glycerin media. Two guinea-pigs inoculated with 0.1 mgm. of third generation culture died from generalised tuberculosis after 64 and 83 days respectively. Two rabbits injected with 1.0 mgm. killed after 123 days: good condition with tuberculous lesions and a few tubercles in lungs.

Eugonic human type.

No. 4.—Six-months cocker spaniel, male. In boarding establishment for three months; on returning home repeated attacks of vomiting, persistent diarrhoea, loss of flesh. Enlarged mesenteric nodes palpated. Exploratory laparotomy showed peritoneal exudate, nodular omental lesions, considerable enlargement of liver and large masses in position of mesenteric nodes. Animal destroyed and mesenteric nodes only received from veterinary surgeon. Enlarged nodes ( $5 \times 3 \times 3.5$  cm.) enveloping intestines for some distance, firm and greyish white on section, no macroscopic caseation; acid-fast organisms numerous in smears. No other macroscopic evidence of tuberculosis reported.

*Histology*.—Extensive necrosis of adipose tissue surrounding enlarged nodes with numerous epithelioid cells and neutrophils; tissue reaction resembles that in tuberculosis in mink. No well-defined tubercles. Acid-fast bacilli very numerous in sections, singly and in groups, especially in necrotic areas; organisms intra- and extra-cellular.

*Bacteriology*.—Strain from guinea-pig inoculated with emulsion of mesenteric nodes; growth slow and poor on egg, no improvement on glycerin egg; faint suggestion of growth on serum and glycerin serum. Two guinea-pigs given 0.1 mgm. of third generation culture died from generalised tuberculosis after 29 and 30 days respectively; two rabbits given 1.0 mgm. died from generalised tuberculosis after 49 and 71 days respectively.

Dysgonic bovine type.

No. 5.—Eighteen-months fox terrier, male. Swelling right side of neck, slowly increasing in size 4 to 6 weeks before admission to clinic. Three months later general condition worse and swelling increased with formation of sinus, discharging small amount of pus. Destroyed.

*Autopsy*.—Enlargement of right mandibular node with sinus opening to right of ventral mid-line of neck; small quantity of greyish pus expressed. Both tonsils enlarged, especially the left. Liver showed numerous greyish foci (2-20 mm.). Mesenteric nodes normal.

*Histology*.—Left tonsil showed area of epithelioid cells with small central zone of necrosis. Numerous epithelioid cell tubercles with central necrosis and secondary infection in right mandibular node. A few small areas of epithelioid cells in left mandibular node. Right tonsil normal. Mesenteric node suspicious of early tuberculosis. No acid-fast organisms in sections.

*Bacteriology*.—Culture from guinea-pig inoculated with emulsion of liver lesions. Grew well on egg and serum; luxuriant growth on glycerin egg and glycerin serum. Two guinea-pigs given 0.1 mgm. of third generation culture died after 79 and 93 days respectively from generalised tuberculosis. Two rabbits killed 125 days after receiving 1.0 mgm. were in good condition with local tuberculous lesions only.

Eugonic human type.

No. 6.—Two-years spaniel, male. Owner's husband died a few months previously of tuberculosis. Loss of condition, occasional cough, temperature 103·5° F. Permission to carry out tuberculin test refused. Animal destroyed.

*Autopsy*.—Tracheo-bronchial nodes enlarged, firm and greyish white ( $5 \times 3 \times 2$  cm.). Diaphragmatic lobe of left lung contained cavity 3 cm. in diameter with a small amount of blood-stained fluid in which occasional acid-fast bacilli were found. No other lesions of tuberculosis.

*Histology*.—Wall of lung cavity consisted of fairly large bronchus lined by granulation tissue containing areas of epithelioid cells. Tracheo-bronchial nodes showed extensive necrosis with periphery of epithelioid cells. Other organs normal.

*Bacteriology*.—Strain from guinea-pig inoculated with emulsion of tracheo-bronchial node. Moderate growth on egg, improved luxuriant growth with faint buff pigmentation on glycerin egg; faint growth on serum, much improved on glycerin serum. Two guinea-pigs inoculated with 0·1 mgm. of third generation culture: one died after 85 days with generalised tuberculosis, the other, killed after 121 days, in good condition but with widespread tuberculosis. Two rabbits given 1·0 mgm. killed after 121 days: both in good condition with local tuberculous lesions only.

Eugonic human type of somewhat reduced virulence.

No. 7.—Five-years setter, male. Nine months' chronic cough. Owner in sanatorium during previous year. Inspiratory dyspnoea; chest dullness, especially on right side. Subcutaneous tuberculin test positive. Animal destroyed.

*Autopsy*.—Lungs emphysematous, tuberculous lesions consisted of a few nodules (2-3 mm.) near root of anterior lobe of left lung. Tracheo-bronchial nodes almost the size of heart with outer wall about 1 cm. in thickness and central cavity containing greyish mucinous fluid. Enlarged nodes had distorted tracheal bifurcation, the right and left main bronchi being at an angle of about 150 degrees. Numerous whitish foci (2-3 mm.) throughout liver.

*Histology*.—Tracheo-bronchial nodes consisted of fibrous tissue bounding hyalinised necrotic areas; no recognisable lymphoid tissue. Liver lesions showed areas of necrosis, bounded by masses of epithelioid cells and traversed by irregular bands of fibrous tissue. Lung lesions showed organising pneumonia; epithelium of bronchioles had almost disappeared; great increase in connective tissue in alveolar walls; several foci of epithelioid cells and numerous giant cells with central nuclei; no necrosis or caseation. No bacilli in sections, but smears of fluid from cystic node showed two beaded acid-fast organisms.

*Bacteriology*.—Culture obtained from guinea-pig inoculated with emulsion of tracheo-bronchial node grew well on egg, moderately well on serum; improvement on glycerin media, faint whitish pigmentation on glycerin egg. Two guinea-pigs inoculated with 0·1 mgm. of third generation culture: one died from intercurrent disease after 19 days, the other after 61 days from generalised tuberculosis. Two rabbits received 1·0 mgm., killed after 117 days: both in fairly good condition, with local tuberculous lesions and retrogressive lesions in lungs.

Eugonic human type.

No. 8.—Three-years fox terrier, male. Malaise, anorexia, temperature 103° F., progressive loss of condition, atrophy of temporal muscles; enlargement of mesenteric nodes palpated. Tuberculin test gave doubtful result; initial temperature high. Mesenteric node enlargement and nodular omental lesions found at exploratory laparotomy. Animal destroyed 14 days after admission.

*Autopsy*.—Omentum thickened and nodular, forming yellowish mass, 1 cm. thick, enveloping intestines. Peritoneal plaque-like lesions (up to 1·5 cm.). Mesenteric nodes fused into single mass ( $7 \times 4 \times 3$  cm.), firm and yellowish white. Portal, gastric and sublumbar nodes enlarged and firm. Several whitish foci (5-10 mm.) in liver.

*Histology*.—Extensive necrosis of mesenteric mass except for thin peripheral zone of epithelioid cells. Liver showed single and conglomerate tubercles throughout (150  $\mu$  to several millimetres). Acid-fast bacilli numerous in mesenteric nodes in sections and smears; fair number in liver lesions.

*Bacteriology*.—Culture from guinea-pig inoculated with emulsion of mesenteric node. Grew poorly on egg, no improvement with glycerin; growth on serum and glycerin serum scanty. Two guinea-pigs died from generalised tuberculosis 36 and 60 days respectively.

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after inoculation of 0.05 mgm. of first generation culture. Two rabbits died from generalised tuberculosis 55 and 70 days respectively after inoculation of 0.5 mgm.

Dysgonic bovine type.

No. 9.—Seven-years terrier, male. Cough for one year, inappetence, diarrhoea, thirst, progressive muscle atrophy and loss of condition. Laboured respiration. Temperature 102.5° F. Destroyed.

*Autopsy.*—400 c.c. yellowish, turbid pleural effusion; 150 c.c. similar fluid in pericardial sac. Pericardium thickened and adherent to lungs, mediastinum, trachea and oesophagus. Liver enlarged but no macroscopic tuberculosis. Two small greyish foci in prostate. Extensive tuberculous myocarditis with greyish nodules (5-15 mm.); endocardium normal. Lungs partially collapsed and no macroscopic tuberculosis; tracheo-bronchial nodes anthracotic. Left kidney two-thirds the size of right; extensive tuberculous pyelitis with caseous areas in medulla and small greyish cortical foci; renal pelvis contained yellowish, mucinous fluid. A few greyish foci in cortex of right kidney and two yellowish areas (8 mm.) ulcerating into renal pelvis. Cortex showed scar of old infarct. Very numerous acid-fast organisms in smears from kidney lesions and a few in pericardial and pleural effusions.

*Histology.*—Prostatic lesions consisted of aggregations of epithelioid cells without necrosis. Only lung lesion found in sections was a small focus of epithelioid cells. Liver contained a few submiliary foci. Myocardial lesions showed extensive central necrosis and cellular infiltration of surrounding muscle. Tracheo-bronchial nodes (macroscopically normal) almost completely necrotic, with only a narrow peripheral cellular zone. Acid-fast organisms numerous in myocardial and kidney lesions.

*Bacteriology.*—Culture direct from kidney lesions. Heavy confluent growth on egg and serum with improved luxuriant growth on glycerin media. Two guinea-pigs inoculated with 0.1 mgm. of second generation culture died from generalised tuberculosis after 71 and 73 days respectively. Two rabbits given 1.0 mgm. killed after 118 days: good condition with local lesions and a few foci in lungs.

Eugonic human type.

No. 10.—Seven-and-a-half-years fox terrier, male. Three days' illness; rapid loss of flesh, increased thirst, albuminuria, dyspnoea for 48 hours, temperature 102.2° F., pulse 168, respiration 64. Liver enlargement palpated. Destroyed.

*Autopsy.*—Single epicardial lesion (1 cm.) left ventricle. Lungs voluminous, innumerable greyish, translucent foci. Nodular omental lesions and peritoneal plaques. Liver enlarged, surface uneven; numerous rounded nodules (2-4 mm.), also larger lesions. Right kidney weighed 25 gm., left 55 gm. Both pelvis distended with yellowish mucinous fluid; caseous medullary lesions and isolated greyish medullary and cortical foci. No macroscopic tuberculous lesions in any lymph nodes. A few acid-fast organisms in smears of lung and liver lesions.

*Histology.*—Submiliary and larger lesions throughout liver; central necrosis even in smallest lesions, and central basophil deposit in many (probably calcium). Kidneys showed glomerular fibrosis, caseous tuberculous pyelitis and calcification of necrotic tubule epithelium. Anthracosis of tracheo-bronchial nodes, no tuberculosis. Lungs showed peribronchial foci of epithelioid cells, and more well-defined tubercles; pulmonary oedema and calcification of much cellular debris within alveoli.

*Bacteriology.*—Organism recovered from guinea-pig passed with material from guinea-pig inoculated with original emulsion of lungs. Good growth on egg and serum, improved luxuriant growth on glycerin media. Two guinea-pigs died from generalised tuberculosis 83 and 96 days respectively after inoculation of 0.1 mgm. of second generation culture. Two rabbits killed 117 days after receiving 1.0 mgm.: good condition with local tuberculous lesions and a few tubercles in lungs and liver.

Eugonic human type.

No. 11.—Two-years terrier, male. One week's history of vomiting, variable appetite and thirst; no loss of condition. Temperature 101.4° F. Enlargement of mesenteric nodes palpated. Destroyed.

*Autopsy.*—200 c.c. blood-stained peritoneal effusion; productive tuberculous peritonitis, including omentum, parietal peritoneum and peritoneal surface of diaphragm. Mesenteric

nodes fused into firm greyish mass ( $12 \times 6 \times 5$  cm.), enveloping several coils of small intestine and with small amount of greyish pus in centre. Liver contained numerous greyish nodules (2-10 mm.) and a few large ones projecting from the surface and with a central depression or "dell". Each kidney showed a small greyish cortical nodule. Several small erosions of mucosa of small intestine in coils enveloped by enlarged mesenteric nodes; lymphoid tissue in intestinal submucosa prominent. A few pleural and epicardial plaques. Bronchial nodes slightly enlarged and anthracotic. Right tonsil showed early epithelioma. Numerous acid-fast organisms in smears from omental lesions.

*Histology*.—Mesenteric nodes almost entirely necrotic. Larger liver lesions showed necrosis. Intestinal erosions not tuberculous, probably due to mechanical interference with circulation by encircling mesenteric nodes. A few acid-fast organisms in liver lesions.

*Bacteriology*.—Culture direct from mesenteric nodes grew well on egg, luxuriantly heaped-up growth on glycerin egg. Two guinea-pigs died from generalised tuberculosis 91 and 93 days respectively after injection of 0.1 mgm. of second generation culture. Two rabbits given 1.0 mgm.: one died after 35 days from intercurrent infection, the other killed in good condition 121 days after inoculation with local tuberculous lesion and a few tubercles in kidney and lungs.

#### Eugonic human type.

No. 12.—Eighteen-months mongrel, male. Fourteen days' illness: increase in rate and depth of respiration, temperature  $103.4^{\circ}$  F. Distemper suspected and serum given. Condition became worse and pleural effusion developed; acid-fast bacilli present. Destroyed.

*Autopsy*.—Poor condition; 500 c.c. straw-coloured pleural effusion. Productive tuberculous pleuritis, including pleural surface of diaphragm. Lungs partially collapsed, oedematous; single macroscopic lesion (2 mm.), subpleural near root of diaphragmatic lobe of right lung; small central hole communicating with cavity, 1 cm. diameter, deep to surface lesion, lined by yellowish grey granular tissue and containing small amount of fluid with numerous acid-fast organisms. Mandibular, mediastinal, tracheo-bronchial and bronchial nodes enlarged and firm.

*Histology*.—Lung lesion consisted of tuberculous granulation tissue surrounded by zone of chronic pneumonia; no necrosis. Mediastinal node showed productive tuberculosis without necrosis. An occasional acid-fast organism in lung cavity. Areas of necrosis and peripheral cellular zone in tracheo-bronchial node.

*Bacteriology*.—Culture direct from mediastinal node grew well on egg and serum and luxuriantly on glycerin media with extensions to water of condensation. Two guinea-pigs inoculated with 0.1 mgm. of second generation culture died from generalised tuberculosis in 64 and 72 days respectively. Two rabbits given 1.0 mgm.: one killed after 124 days in good condition, with local tuberculous lesion and a few old tubercles in the lungs, the other killed after nine days because of injury.

#### Eugonic human type.

No. 13.—Three-and-a-half-years fox terrier, male. Three weeks' history, loss of condition, thirst; slight albuminuria, enlarged mesenteric nodes palpated. Reaction to tuberculin test doubtful. Exploratory laparotomy revealed nodular lesions in omentum. Destroyed.

*Autopsy*.—Lungs oedematous with a few greyish, translucent foci (2-3 mm.). Tracheo-bronchial nodes macroscopically normal. Mesenteric nodes converted into three masses of firm yellowish white tissue ( $8 \times 5 \times 3$ ,  $5 \times 3 \times 2$ ,  $3 \times 2 \times 2$  cm.) covered with thickened omentum and adherent to several coils of intestine. Plaques on peritoneal surface of diaphragm. Sublumbar node enlarged and firm ( $3 \times 2 \times 1$  cm.). Spleen contained one whitish nodule (3 mm.). Multiple rounded lesions in liver (2-10 mm.), mostly in portal region. A few acid-fast organisms in smears of liver lesions.

*Histology*.—Extensive necrosis in mesenteric masses. Productive lesions, large and small, in liver, the former with central necrosis. A few epithelioid cell tubercles in lungs. Spleen contained two foci with central necrosis. No acid-fast organisms seen in sections.

*Bacteriology*.—Culture direct from liver lesions grew well on egg and serum, more luxuriantly on glycerin media with extension to water of condensation. Two guinea-pigs inoculated with 0.1 mgm. of second generation culture: one died after 34 days from intercurrent infection and showed early tuberculosis, the second died after 63 days with wide-

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spread tuberculosis. Two rabbits inoculated with 1·0 mgm. killed after 123 days in good condition, with local lesions and a few old tubercles in lungs.

Eugonic human type.

No. 14.—Ten-years collie, male. Ten days' illness; vomiting, diarrhoea, inappetence and loss of condition. Temperature 104·2° F. Enlarged mesenteric nodes palpated. Destroyed.

*Autopsy*.—Omentum and mesentery studded with firm nodular lesions. Lungs and thoracic lymph nodes free from tuberculosis. Liver contained about a dozen firm, yellowish nodules. Mesenteric nodes formed a firm yellowish white mass ( $7 \times 4 \times 3$  cm.) adherent to several coils of small intestine and colon, which were surrounded by thickened nodular omentum; nodular lesions (2-30 mm.) in mesentery. A few acid-fast bacilli in smears of liver lesions.

*Histology*.—Chronic passive congestion of liver and conglomerate productive tubercles; mesenteric nodes almost entirely necrotic.

*Bacteriology*.—Culture from spleen of guinea-pig injected with emulsion of liver lesions grew well on egg and serum, more luxuriantly with secondary heaped-up colonies on glycerin media; slight yellowish pigmentation on glycerin egg. Two guinea pigs inoculated with 0·1 mgm. of first generation culture: one died from intercurrent infection, the other from generalised tuberculosis after 54 days. Two rabbits killed 123 days after injection with 1·0 mgm. were in good condition, with local lesions and a few retrogressive tubercles in lungs.

Eugonic human type.

No. 15.—Nine-and-a-half-years Pomeranian, male. One week's history of laboured, shallow respiration, very thin, increased thirst. Destroyed.

*Autopsy*.—Lungs voluminous, studded with small greyish, translucent foci, aggregated in places to form areas up to 1 cm. in diameter; bronchi contained yellowish exudate. No macroscopic liver lesions. Myocardium riddled with lesions (4-10 mm.), more numerous on right side. Renal cortex of both kidneys showed numerous 2-4 mm. greyish foci. Mediastinal nodes enlarged and firm. Tracheo-bronchial node enlarged, anthracotic ( $4\cdot5 \times 2 \times 2$  cm.). Acid-fast organisms in groups in smears from lung lesions.

*Histology*.—Liver simulated lymphadenosis, the periportal connective tissue being infiltrated with cells, mainly epithelioid in type without tubercle formation or necrosis. Numerous tubercles with and without central necrosis in tracheo-bronchial node. Larger lung lesions consisted of areas of consolidation, alveoli filled with epithelioid cells, lymphocytes and plasma cells. Numerous acid-fast organisms in all lesions, especially within epithelioid cells in liver.

*Bacteriology*.—Culture direct from lung lesions gave good confluent growth on egg and serum, improved luxuriant growth on glycerin media. Two guinea-pigs inoculated with 0·2 mgm. of second generation culture: one died during very cold weather after 105 days and had widespread tuberculosis; the other, killed after 121 days, showed similar lesions. A guinea-pig injected with the original material, killed after 122 days, showed similar widespread chronic tuberculosis. Two rabbits killed 121 days after inoculation of 2·0 mgm. of culture were in good condition, with local tuberculous lesions and a few old tubercles in lungs.

Eugonic human type of reduced virulence.

No. 16.—Three-years fox terrier, male. Three months' history of dullness and loss of flesh. Tuberculin test positive (104° F. after two hours, initial temperature 101·2° F.). Destroyed.

*Autopsy*.—150 c.c. straw-coloured pleural effusion. Pleural plaques, especially over diaphragm; lungs showed numerous minute greyish, translucent foci. A small subpleural lesion in cardiac lobe of right lung was possibly ulcerating through pleura. Liver contained innumerable firm whitish foci (2-12 mm.). Both kidneys showed a few cortical nodules (3-5 mm.); mediastinal nodes enlarged to form firm mass surrounding base of heart and adherent to lung, pleura and diaphragm. Tracheo-bronchial node measured  $2 \times 1 \times 1$  cm. and was firm and greyish. One mesenteric node enlarged to 1 cm. diameter; portal and gastric nodes enlarged, firm and adherent to stomach and head of pancreas.

*Histology*.—Lungs contained fairly large tubercles with central necrosis. Liver showed

miliary and submiliary tubercles and large conglomerate lesions with central necrosis. Pleural lesions productive in type, without necrosis. Mesenteric, portal and mediastinal nodes showed very extensive necrosis with peripheral zone of epithelioid cells.

*Bacteriology.*—Culture direct from mediastinal node; heavy confluent growth on egg and serum, luxuriant growth with secondary colonies and extension to water of condensation on glycerin media. Two guinea-pigs injected with 0·1 mgm. of second generation culture: one died from intercurrent infection but showed extensive tuberculosis, the other died from generalised tuberculosis after 49 days. Two rabbits injected with 1·0 mgm. were killed after 112 days, in good condition with local lesions and a few retrogressive tubercles in lungs.

Eugonic human type.

No. 17.—One-year fox terrier, female. Three months' history; cough, loss of flesh, ravenous appetite until fourteen days before admission, occasional vomiting and slight respiratory distress. Circumscribed enlargement palpated left side of pelvic region. Tuberculin test gave doubtful reaction—102·8° F. rising to 103·8° F. at fourth hour. Destroyed.

*Autopsy.*—Emaciated; small amount of greenish fluid in peritoneal cavity. Lesions in myocardium comprising single nodule 1 cm. in diameter at junction of right auricle and ventricle and numerous 2-3 mm. lesions elsewhere. Both lungs heavily studded with small greyish, translucent tubercles, most closely aggregated in anterior lobe of right lung. Liver enlarged, numerous firm yellowish lesions 5-8 mm. in diameter and innumerable minute lesions in intervening tissue. Spleen enlarged with innumerable greyish foci resembling hyperplastic follicles on section. Enlarged lymph node near head of pancreas and firm, whitish lesion affecting the latter for an area of 2 x 2 cm. Omentum contained numerous firm greyish nodules. Left kidney hydronephrotic due to obstruction of ureter near entry into bladder by a mass of firm greyish tissue (3 x 2 x 2 cm.), cortex of both kidneys contained a few 2-3 mm. greyish foci. Mediastinal nodes not recognisable, being replaced by a mass of tissue adherent to pericardium and anterior lobe of right lung. Tracheo-bronchial, mandibular, axillary and suprasternal nodes slightly enlarged. Acid-fast bacilli fairly numerous in smears of liver, lung and myocardial lesions.

*Histology.*—Lung lesions consisted of epithelioid tubercles, and, in anterior lobe of right lung, a diffuse cellular pneumonia; considerable pulmonary oedema. Splenic foci consisted of miliary epithelioid cell tubercles. Mediastinal mass largely necrotic, with peripheral zone of epithelioid cells and central basophil material, probably calcium. In the liver there remained only about one-third of the parenchyma, the remainder of the tissue consisting of single and conglomerate tubercles. Lesions in myocardium and enveloping left ureter were productive in character, with little necrosis.

*Bacteriology.*—Culture from lung tissue by direct methods, grew well as confluent growth on egg and serum and more luxuriantly with heaped-up secondary colonies on glycerin media. Guinea-pig inoculated with original material killed with moderate degree of tuberculosis 121 days later. Two guinea-pigs inoculated with 0·1 mgm. of second generation culture: one died from intercurrent infection, the other from generalised tuberculosis after 72 days. Two rabbits given 1·0 mgm. were killed after 114 days: both in good condition with local lesions and a few retrogressive tubercles in the lungs.

Eugonic human type of moderate virulence.

No. 18.—Four-years mongrel, male. Swelling behind angle of right mandible for six weeks; pain on pressure; temperature 103° F. Small portion of enlarged mandibular node removed at biopsy diagnosed as tuberculous. Subcutaneous tuberculin test could not be employed owing to high initial temperature; no reaction obtained with intradermal and von Pirquet tests. Condition now much worse, pleurisy with effusion. Destroyed.

*Autopsy.*—Good bodily condition, 500 c.c. pleural effusion and extensive productive tuberculous pleurisy. Lungs showed innumerable greyish foci throughout; tracheo-bronchial nodes not macroscopically enlarged. Suprasternal nodes measured 3 x 2 x 1 cm. and consisted of firm greyish tissue. Right lateral lobe of liver converted into spherical cyst, 7 cm. in diameter, containing clear fluid with whitish flocculi. Small nodules, 2-3 mm. in diameter, scattered throughout liver. Gastric and portal nodes formed a mass measuring 3 x 2 x 1 cm.; omentum studded with firm whitish, nodular lesions. Serous

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covering of both faces of diaphragm covered with firm plaques. Numerous acid-fast bacilli in smears of large liver lesion. Several greyish foci in both kidneys, renal pelvis contained brownish calculi.

*Histology.*—One lung tubercle showed extension through overlying pleura. Tracheo-bronchial node tuberculous with extensive necrosis; mediastinal node similar, with basophil granules in centre of necrotic areas. Suprasternal and pharyngeal nodes resembled tracheo-bronchial. Occasional acid-fast bacilli seen in sections.

*Bacteriology.*—Strain isolated direct from mediastinal node, produced confluent growth on egg and serum with greatly improved growth on glycerin media. Three guinea-pigs inoculated, one with original material and two with 0·1 mgm. of second generation culture: all died during very cold weather, showing some degree of progressive tuberculosis. Two rabbits killed 114 days after the injection of 1·0 mgm.: both in good condition with local lesions and a few retrogressive tubercles in lungs.

Eugonic human type.

No. 19.—Three-years terrier, female. Nine months' illness; loss of condition, occasional vomiting, repeated attacks of diarrhoea, coughing. Temperature 102·3° F. Destroyed.

*Autopsy.*—Carcass emaciated, 250 c.c. fluid in pericardial sac. Pericardium and myocardium were the seat of extensive tuberculous lesions. Lungs showed innumerable greyish foci throughout. All thoracic nodes slightly enlarged. Two lesions in muscularis of small intestine, 6 and 9 cm. respectively from pylorus. Liver contained irregular whitish lesions, especially numerous in portal region. Mesenteric nodes macroscopically normal, gastric and hepatic measured 2 x 1 x 1 cm., firm and yellowish. Left kidney showed greyish infarct-like lesion in cortex; right kidney several 2-3 mm. cortical greyish foci. Numerous acid-fast organisms in smears of mediastinal node.

*Histology.*—Intestinal lesions limited to muscularis and subserosa. Lung foci accompanied by considerable consolidation of adjacent alveoli, exudate consisting mainly of epithelioid cells.

*Bacteriology.*—Culture obtained direct from mediastinal node. Heavy confluent growth on egg and serum with heaped-up improved growth on glycerin media. Two guinea-pigs inoculated with 0·1 mgm. of second generation culture: one died after 22 days from intercurrent disease but with widespread tuberculosis, the other after 89 days with generalised tuberculosis. Two rabbits given 1·0 mgm.: one died from an intercurrent disease after 37 days, the other killed after 129 days in good condition with local tuberculous lesion and a few old tubercles in lungs.

Eugonic human type.

No. 20.—Three-years Sealyham, male. Three to four months' history of chronic cough, variable appetite, loss of flesh and diarrhoea. Temperature 103° F. Enlargement of mesenteric nodes palpated. Destroyed.

*Autopsy.*—Firm whitish nodules throughout mesentery. Mesenteric nodes measured 6 x 4 x 4 cm. and adherent to several loops of intestine. Liver contained innumerable lesions varying in size from 2 to 20 mm. Single small greyish nodule in cortex of left kidney. Lungs showed miliary greyish foci evenly distributed throughout. Tracheo-bronchial nodes enlarged and spherical; mediastinal nodes enlarged and firm (6 x 2 x 2 cm.). Occasional acid-fast bacilli in smears of liver lesions.

*Histology.*—Affected lymph nodes showed extensive necrosis, with no obvious difference between tracheo-bronchial and mesenteric. In addition to liver lesions above described, there were periportal foci of epithelioid cells. Lung lesions showed no central necrosis.

*Bacteriology.*—Culture obtained direct from ground-up liver tissue and mesenteric node. Growth on egg and serum slow and poor with no improvement on glycerin media. Two guinea-pigs died from generalised tuberculosis 31 and 38 days after injection of 0·1 mgm. of second generation culture, and two rabbits 41 and 48 days after injection of 1·0 mgm.

Dysgonic bovine type.

No. 21.—Five-years Airedale, female. Indefinite history of about two weeks; attempts at vomiting, inappetence, loss of flesh, diarrhoea and laboured respiration. Pleural effusion, atrophy of temporal muscles. Temperature 102·8° F. Destroyed.

*Autopsy.*—Pleural cavities contained two litres of haemorrhagic effusion; parietal pleura showed productive tuberculous lesions. Mediastinal and tracheo-bronchial nodes enlarged,

the latter measuring  $6 \times 4 \times 3$  cm. Both lungs showed greyish, translucent foci (3-5 mm.) near hilum. No enlargement of any of the abdominal nodes.

*Histology*.—In addition to circumscribed lesions with central necrosis, the lung showed a diffuse consolidation in the tissue between the focal lesions, cells being mainly epithelioid in type. Tracheo-bronchial and mediastinal nodes almost entirely necrotic. Liver showed central lobular fatty changes and submiliary foci with necrotic centre. A few acid-fast organisms seen in lung lesions and large anaerobic organisms in liver and lung (carcass examined two days after death).

*Bacteriology*.—Culture obtained direct from tracheo-bronchial node. Heavy confluent growth on egg and serum, improved luxuriant growth with heaped-up secondary colonies on glycerin media. Two guinea-pigs inoculated with 0.2 mgm. of second generation culture: one died after 16 days from intercurrent disease and some degree of tuberculosis, the other after 92 days from generalised tuberculosis. Two rabbits injected with 2.0 mgm., killed after 148 days, in good condition with local tuberculous lesions and a few old tubercles in lungs.

Eugonic human type.

No. 22.—Twelve-years Alsatian, male. Six weeks' history of thirst, inappetence and occasional vomiting. Destroyed.

*Autopsy*.—Tuberculous peritonitis and pleuritis without effusion. Nodular lesions of omentum and mesentery. Duodenum showed an ulcer 2 cm. from pylorus; haemorrhage had recently occurred and the whole of the intestinal contents contained blood. Cortex and medulla of right kidney contained greyish nodules, and small lesions were present on peritoneal covering of both kidneys. Lungs free from macroscopic lesions. All thoracic nodes enlarged and mainly composed of greyish nodules, 3-4 mm. in diameter, containing a material resembling toothpaste. Liver contained a large number of lesions of similar appearance. Lesions of this kind have not previously been observed by us in tuberculosis in the dog. The chalky material was slightly gritty, and no acid-fast bacilli were found in smears prepared from it, although several were seen in preparations from the periphery of the liver lesions.

*Histology*.—Duodenal ulcer not tuberculous but may have been caused by pressure from necrotic tuberculous foci in subserosa. All tuberculous lesions showed considerable amount of basophil deposit in the centre and a narrow peripheral cellular zone. A few submiliary lesions were found in the lung, with occasional acid-fast organisms. In the kidney, acid-fast bacilli were especially numerous in the medullary lesions; tubercle bacilli could be seen (even under the low-power objective) aggregated into huge masses in the region of the pelvis.

*Bacteriology*.—Culture obtained direct from an omental nodule. Heavy growth on egg and serum, improved luxuriant growth on glycerin media, with yellowish-pink pigment on glycerin egg. Two guinea-pigs inoculated with 0.2 mgm. of a second generation culture: one died from intercurrent disease and the other after 71 days with generalised tuberculosis. Two rabbits inoculated with 2.0 mgm., killed after 119 days, in good condition with a local tuberculous lesion only.

Eugonic human type.

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## TREATMENT OF TUBERCULOSIS OF THE LARYNX BY INJECTION

BY LIEUT.-COLONEL SIR JAMES R. ROBERTS,  
C.I.E., M.B., M.S., F.R.C.S.  
Dewas Senior, Central India.

JUDGING from a recent paper by Scott Stevenson and Heaf, the treatment both intrinsic and extrinsic of laryngeal tuberculosis still remains a difficulty. I wish to describe here a method of dealing with this condition which we are using in India. It is one which is very applicable to patients in this country, for it is simple, cheap, easy to administer, and therefore suitable for Indian sufferers. It is used also for chronic laryngitis and pharyngitis with marked success.

It is our experience that a majority of patients who come here for treatment of pulmonary tuberculosis suffer from severe cough associated with a certain amount of vocal failure; this distressing and constant cough leads to sleeplessness, and the combined evils usually call for considerable resource in treatment. Examination with the laryngoscope shows an intense red irritated pharynx and larynx, not necessarily associated with ulceration, which produces both dysphonia and dysphagia. Both groups are treated similarly by the method described below. The results are usually remarkable and deserve to be better known, as the alleviation to the sufferers is so great.

*Technique:* The procedure is that of injecting the interior of the larynx, the needle delivering a dose of 1 in 60 carbolic solution underneath the mucous membrane, where it diffuses into the tissues around. Half a c.c. is the usual dose.

The solution I advocate for injecting the larynx is:

R Acid carbolic  $\frac{1}{2}$  drachm.  
Glycerine 2 drachms.  
Sterile water, 4 ounces.

Planocaine can be added as required; a separate solution of this is drawn on when necessary, as prolonged exposure of planocaine to strong carbolic seems to take the edge off this local anaesthetic.

The patient is placed on his back with a pillow or roll under his neck, in order to make the larynx prominent. The skin over the larynx is first cleaned thoroughly, dried, and painted with tincture of iodine. With aseptic precautions a fine needle,  $\frac{1}{4}$  inch long, No. 16 continental gauge, is then attached to a 2 c.c. syringe, which is held in the right hand like a pen. The left forefinger is then applied to the skin over the thyroid cartilage and the notch defined by touch, with the hyoid bone felt higher up. The upper part of the notch is located and the needle introduced here through the skin, and advanced a little deeper into the subcutaneous tissue; then the whole syringe is raised until it is almost in contact with the side of the lower jaw. This brings the needle behind the margin of the ala of the thyroid cartilage on one side directed downwards and obliquely backwards, and it is then inserted, following the inner surface of the cartilage, until it has penetrated about half an inch, its point resting in the submucous tissue in the proximity of the ventricle of the larynx. The dose is delivered by pressing down the piston rod, and the needle and syringe quickly removed. It is rarely that any cough is raised. A practised hand does the procedure rapidly.

The patient is previously prepared by instruction not to swallow, not to cough, and to breathe normally. If he swallows he pulls up the larynx, bends the needle, and is apt to cause it to pierce the mucous membrane; the dose is thus delivered inside the larynx and a most explosive cough is started. To prevent coughing it is as well to ask him to clear his bronchi of sputum before injecting. Never employ a brittle needle lest it break in the larynx. The needle must be very sharp, the point and sides of the bevel ground so that it cuts into the skin and tissues. It is important to keep an Arkansas stone slip on which to sharpen the needle and to grind the level flat, obliterating the curve with which the manufacturers turn out needle points.

The large larynx with a prominent Adam's apple in some males is easily injected; the smaller one in females is a little difficult, and the notch sometimes not easy to define. This difficulty can be avoided by beginning exactly in the space between the hyoid and thyroid. Remember that the thyroid ala is thicker than one supposes, and beginners are apt to strike the upper edge of the ala; to avoid this go farther in after penetrating the skin. The other side of the larynx is injected as a rule after twenty-four or forty-eight hours. As improvement takes place the interval can be increased to seventy-two hours. After the second injection the interval between can be increased, or otherwise a swelling may be formed which embarrasses the breathing.

To deal with manifestations in the extrinsic portions of the larynx, the arytenoids, epiglottis and ventricular fold, another and easy method is to cocaineise the throat and introduce the left forefinger down the throat until it rests on the epiglottis, then, placing the right forefinger on the skin above the hyoid to gauge the space between the two fingers. Then with the right hand insert the needle above the hyoid bone, and pass it through the skin to the base of the tongue; some of the solution is then injected at the junction of the epiglottis and tongue, and some on either side without piercing the mucous membranes. The finger in the throat acts as a guide.

This treatment does not require more than a few injections. The pain diminishes rapidly and the progress towards recovery is often striking. The cough is relieved, he can sleep again, he can eat with comfort, and if the operator adds some novocaine to the solution at the time of injection the relief is of course more pronounced.

In Kashmir in 1938, while I was demonstrating the method to a number of doctors in the Mission Hospital, a young man was brought into the clinic on a wooden cot. In appearance he was an emaciated wreck, with only a low whisper for a voice. He complained of a marked and painful dysphagia and cough. I injected one side of the larynx, then the base of the tongue, and told his friends to bring him back in forty-eight hours. By then he was already much relieved and able to eat freely. There were ulcers about the base of the epiglottis, and the intra-arytenoid space was also involved. The lesion in the lung was not severe. On his third visit in seventy-two hours he walked into the clinic, and four weeks later he insisted on going home to a nearby village.

On another occasion a man of about forty came into the clinic only just able to whisper on account of chronic laryngitis produced by catarrhs and using his voice to address audiences mostly out of doors. He explained he had been round India trying to get a cure, and had failed. After six injections at weekly intervals, his voice was full and clear. When I asked

him how he came to lose his voice, he replied, "I am a public agitator: to lose my voice is to fail in my profession!"

The method I have described is based on a principle already evident in treating mucous membranes, that surface applications do not affect disease deep to mucous membranes. This is illustrated by the treatment of tuberculosis of the larynx, the ulcerated cases showing rapid healing when injected outside, or rather below the surface. It is shown with equal success in tuberculosis of the bladder, and in ulceration of the rectum, whether due to T.B. or proctitis or dysentery.

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## REPORTS OF SOCIETIES

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### ANNUAL GENERAL MEETING OF THE NATIONAL ASSOCIATION FOR THE PREVENTION OF TUBERCULOSIS

THE Annual General Meeting of the National Association for the Prevention of Tuberculosis was held in Tavistock House on April 18, 1940. The chair was taken by the Marchioness of Titchfield, who, moving the adoption of the Annual Report, referred to the part played at the beginning of the war by the Association, when the deputation waited on the Minister of Health. The views of the tuberculosis movement had been kept prominently before the authorities. The Christmas Seal Sale was a source of satisfaction and profit, not only to the Association, but to its affiliated Care Committees throughout the country.

Formal business was followed by an address by Sir Hugh Walpole.

He dealt with the effect of sickness on the character. Sickness and pain for the time being do not seem to improve the character, for the sick person is conscious of a frightening sense of separation. His friends must then make the effort to cross over from their own world, the world of safety, into the other dark world of danger. One must be constantly aware of the loneliness, despair and unhappiness of the sick.

The two experiences from which he personally had learned most were his part in the last war and his severe illness. Life is intended to be hazardous; there is no danger which is not meant for us to face, and an easy, safe life teaches us practically nothing. We must have experience of the world of danger to complete our experience—not only that, we must encourage our contact with it. For sickness and pain do not seem to be sickness and pain when they are mingled with one's own experience. Any one of us may at any moment be launched into that other world, perhaps the better and finer of the two, and absolutely necessary to every human being's complete experience.

The address was followed by a discussion on "Tuberculosis Care Work in War-time", opened by Dr. J. B. McDougall.

## CORRESPONDENCE

## MASS RADIOGRAPHY OF THE CHEST

*To the Editor.*

DEAR SIR,

We have read with interest the article by Mr. P. G. Sutton, "Mass Radiography of the Chest, with Special Reference to the Pulmograph", published in the journal, Vol. XXXIV., No. 2, April, 1940. Owing to the great importance we attach to this work in the control of pulmonary tuberculosis, we wish to offer the following criticism:

It is evident that Mr. Sutton is working on the same assumption as Dr. A. Robinson Thomas, who published an article in *Public Health*, September, 1939, and that is that it is necessary and even desirable to produce a plant for mass radiography of the chest at a low cost. This was dealt with at some length in correspondence published in *Public Health*, January, 1940.

Briefly the position is this. Mass radiography offers so far the only hope of eventual control of pulmonary tuberculosis, and, while the authors view with enthusiasm any attempt by manufacturers to overcome certain technical difficulties, they feel that a low-priced plant (the quality of the pictures must suffer) may lead to these being installed in many X-ray departments as a mere novelty, and not for serious mass radiography, and so the method would in time fall into disrepute.

If serious mass radiography is to be attempted by a public body, and they are satisfied with the diagnostic quality of test films, £5,000 is very little to pay for such a plant.

We wish to draw your attention to a statement in Mr. Sutton's article, p. 57: "The most rapid lens in existence at the present time has an aperture of f/0.85, but such lenses are extremely expensive and are not suitable for X-ray screen photography." Referring to the *Lancet*, June 10, 1939, and November 11, 1939, the f/0.85 was made by Messrs. Zeiss for Mr. Collender especially for miniature radiography, and had it been marketed the cost would have been about £300. We have personally also used a Sonnar f/1.5, but, although results are obtainable, there is no comparison at all with the truly wonderful result obtained with the f/0.85. The extra speed of the lens also decreases the power necessary to produce results. We further suggest that the tube current of 120 ma. is inadequate to deal with all cases, and further, when large numbers of people strange to the procedure are X-rayed with an exposure of 0.2 to 0.3 second there is certain to be movement in a good number of them. We suggest that 0.1 second should, if possible, be the maximum time and this would require up to 500 ma., so a four-valve plant and rotating anode tube seem to be indicated.

We believe that there is an Eastman XXX film in the United States of America corresponding to the Agfa fluoroid (H. and D. 27,000), though we have been unable to obtain it here.

We think that the manufacturers are to be congratulated on their method of counter-balancing the camera-screen tunnel and tube head; from the illustration it seems simple but effective. Further particulars of the method of photographing the record card would be of interest, and we cannot but regret that we have seen no samples of the miniature X-rays produced.

With regard to projection, we have found that a good quality projector taking Kodas slides or 35 mm. strips is suitable, and an enlargement to about 12 by 15 inches is the maximum before grain is apparent. Of course fine grain developer is a *sine qua non*.

We would like to endorse the view expressed by Mr. Sutton that the greater the number of mass radiographs taken in the day the greater must be the number and efficiency of the staff organising records.

Yours faithfully,

B. A. DORMER (*Medical Superintendent*).  
M. GIBSON (*Radiographer*).

KING GEORGE V JUBILEE HOSPITAL FOR TUBERCULOSIS, DURBAN.

June 10, 1940.

## REVIEWS OF NEW BOOKS

*Artificial Pneumothorax. Its Practical Application in the Treatment of Pulmonary Tuberculosis.* Edited by EDWARD N. PACKARD, JOHN N. HAYES, and SIDNEY F. BLANCHET. London: Henry Kimpton. Price 21s.

This excellent volume is a conjoint work by physicians attached to the Trudeau Foundation at Saranac Lake. It is well produced and ably written, and compasses within a reasonable space of some three hundred pages a well-balanced and up-to-date account of artificial pneumothorax treatment. It includes references to the more important literature, and at the same time gives much of the personal experience of the authors, an experience which is extensive and varied. It is thus not only a textbook on the subject, but also a volume of reference to the best American opinion which should be widely read.

*Silicosis.* Proceedings of the International Conference held in Geneva from August 29 to September 9, 1938. International Labour Office: P. S. King. Price 5s.

This volume comprises the reports and discussions at the round-table conference on silicosis sponsored by the International Labour Office in 1938. It thus gives a detailed review of the advances in knowledge on this subject since the conference which met in Johannesburg in 1930. Such reports almost always suffer from the difficulty in collating the material presented and in giving to readers any idea of the conclusions to which each discussion led; and unfortunately this report is no exception to the rule. It is thus a book for the expert. For him it represents a mine of information covering a wide field of reading that has here been collected together in successive brief reports and discussions.

*St. Thomas's Hospital Reports.* Second Series, Vol. IV, 1939.

The current volume of the St. Thomas's Hospital Reports well maintains the consistently high standard that has been set in previous years, and the hospital is to be warmly congratulated on its achievement. It is a beautifully produced volume, with articles ranging over a wide field of interest, ably written and informative. The papers of special interest to those occupied with thoracic medicine include one on oxygen therapy, reviewing modern methods of administration; another on empyema thoracis, dealing mainly with its management; and a most interesting paper on the physiology of respiration, referring more particularly to asphyxia neonatorum and its treatment.

## OBITUARY

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### EVELYN MARY HOLMES

We deeply regret to announce the death of Evelyn Mary Holmes on September 4 last after a brief illness, at the early age of 41. For years she had struggled against ill health, and in spite of it had done a great deal of valuable work in the field of tuberculosis, holding a number of important appointments in which she gained a wide esteem, not only for her outstanding ability but also for her personal charm and kindness. The importance of her published work was acknowledged when she was given her M.D. last year in the University of Manchester. She travelled widely, studying in Canada, Denmark, and Sweden.

All who knew her will regret the loss of one so much imbued with the spirit of healing, and wish to extend to her family sincere condolences.

C. H.

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